A Dissertation on

OCULAR MANIFESTATIONS IN PATIENTS WITH CHRONIC KIDNEY DISEASE – A HOSPITAL BASED STUDY

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CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr. P.SHOBHA** on "**OCULAR MANIFESTATIONS IN PATIENTS WITH CHRONIC KIDNEY DISEASE – A HOSPITAL BASED STUDY** " during her M.S.(Ophthalmology) course from June 2010 to April 2012 at the Government Stanley Medical College & Hospital, Chennai .

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DECLARATION

I , hereby declare that this dissertation entitled "**OCULAR MANIFESTATIONS IN PATIENTS WITH CHRONIC KIDNEY DISEASE – A HOSPITAL BASED STUDY"** is a bonafide genuine research work carried out by me under the guidance of **Prof. Dr. K.Basker,** M.S.,D.O., HOD, Department of Ophthalmology , Government Stanley Medical College and Hospital, Chennai – 600001.

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CONTENTS

PART - I

INTRODUCTION

Chronic kidney disease (CKD) is a worldwide health problem. There is a rising incidence of renal failure due to chronic kidney disease and this phenomenon is common in both the developed and under developed countries. There is a significant mortality and morbidity associated with this condition and it drastically reduces the quality of the patient's life.

Normal functions of the kidneys can be affected by a variety of diseases and medical conditions. These cause a reduction in GFR, metabolic imbalances and retention of harmful waste products. A majority of patients progress to end stage kidney disease and may require dialysis or renal transplantation.

Chronic kidney disease leads to a lot of systemic effects that affects a variety of systems in the body. The eye also shows changes due to long standing kidney disease. Some systemic diseases such as diabetes, hypertension and auto immune disorders affect the kidneys as well as the eye. Ocular manifestations may arise as a result of the primary diseases causing renal failure or as a result of the secondary effects of renal failure itself. It is thus very difficult to ascertain whether the systemic effects are due to the disease which caused the renal failure or secondary to the

changes caused by the kidney disease unless the patient is monitored continuously throughout the course of the disease.

CHRONIC KIDNEY DISEASE

Definition

The National Kidney Foundation (NKF) Kidney Disease Outcome Quality Initiative (K/DOQI) Work Group set about to develop clinical practice guidelines to define chronic kidney disease (CKD) and to classify stages in its progression.

CKD was thus defined as the presence of kidney damage or decreased level of kidney function for three months or more, irrespective of diagnosis.

Calculation of Glomerular Filtration Rate (GFR)

Glomerular Filtration rate is an important element in making a diagnosis of chronic kidney disease as well as to assess the progression of the disease. Creatinine levels cannot serve as an estimate or indicator of the filtering ability of the kidney as it is both filtered in the glomerulus as well as secreted by the proximal tubule. Creatinine clearance also fails as an indicator of Glomerular filtration rate as it is known to overestimate GFR by around 40% in normal individuals.

Classic methods for measurements of GFR, including the gold-standard inulin clearance, are cumbersome and are not clinically feasible. In adults, the normal GFR based on inulin clearance and adjusted to a standard body surface area of 1.73 m^2 is 127 ml / min / 1.73 m^2 for men and 118 ml / min / 1.73 m^2

for women (with a standard deviation of approximately 20 ml / min / 1.73 m^2). After age 30, the average decrease in GFR is 1 ml / min / 1.73 m^2 per year.

Equations based on serum creatinine but factored for gender, age, and ethnicity are the best alternative for estimation of GFR. The most commonly used formula is the **Cockcroft-Gault equation**[20]. This equation was developed to predict CrCl, but has been used to estimate GFR.

CrCl = [(140-age) x Weight (Kg)]/[S. Cr x 72] [x 0.85 in women]

The Cockcroft-Gault formula is reasonably accurate in mild renal impairment with a GFR of around 50 ml/min but can overestimate GFR by up to 100 per cent when GFR is less than 10 ml/min.

The abbreviated **MDRD equation**[21,22] is recommended for routine use and requires only serum creatinine, age, gender and race.

GFR = 186.3 x (S. Cr)-1.154 x (age)-0.203 x (0.742 if female) x (1.21 if black). This formula is more accurate especially at low clearance and in preferred to the Cockcroft-Gault method.

Stages of CKD

The Kidney Disease Outcomes Quality Initiative (K/ DOQI) guidelines have classified chronic kidney diseases into five stages.

Stage 1 : Normal or increased glomerular filtration rate (GFR) but some evidence of kidney damage reflected by microalbuminuria / proteinuria , hematuria or histological changes.

Stage 2 : Kidney damage with a mild decrease in GFR (60 -89 ml/min/1.73m^2).

Stage 3 : Moderate decrease in GFR (30 – 59 ml/min/1.73m^2).

Stage 4 : Severe decrease in GFR (15 – 29 ml/min/1.73m^2).

Stage 5 : Is when renal replacement therapy in the form of dialysis or transplantation has to be considered to sustain life.

EPIDEMIOLOGY OF CHRONIC KIDNEY DISEASE IN INDIA

The exact data of the prevalence of chronic kidney disease in India is not widely available. The first published study was from Apollo Hospital, Chennai by Dr. M. K. Mani[24]. It was a population based study, primarily at preventing kidney disease particularly by detection and treatment of diabetes and hypertension Prevalence of CKD in the surveyed community was 0.16%.

Another study was from All India Institute of Medical Sciences by Dr. Sanjay Agarwal[23]. This was a community based study done in urban Delhi where 4972 subjects were screened with blood urea and serum creatinine estimation with a specific aim to find out the prevalence of CKD. The prevalence of CKD,was defined as serum creatinine > 1.8mg% persisting for more than 3 months in the absence of any reversible factor.CKD prevalence was calculaled to be 0.79% or 7852 per million population

Jha and Modi [26] reported a study from Bhopal from 2002-2005. They reported that the average crude and age adjusted incidence rates of End stage renal disease were 151 and 232 per million population respectively.

A group of nephrologists from Indian Society Of Nephrology started a project to analyse the data on chronic kidney disease patients, called Chronic Kidney Disease Registry Of India .

Screening and Early Evaluation of Kidney disease (SEEK) was started in 2006 by nephrologists from USA and India.This study reported a very high prevalence of 17.4% of CKD in the participants.

The exact burden of chronic kidney disease in India is not known. Studies have shown that the prevalence of chronic kidney disease ranges from 0.78% to 17.4% while the incidence is 151 per million population.

ETIOLOGY OF CHRONIC KIDNEY DISEASE

Renal insufficiency can ensue from a primary renal disease or a systemic disease which affects the kidneys. Chronic systemic diseases like diabetes mellitus and hypertension are some of the common causes of chronic kidney disease. The slow progression of these systemic diseases make it more difficult to identify the onset of kidney dysfunction. These are the preventable causes of renal failure. Thus continuous and rigorous monitoring of patients with such systemic diseases is essential for the early detection of involvement of kidneys.

CAUSES OF CHRONIC KIDNEY DISEASE [1]

Most Common causes

Diabetes mellitus

Hypertension

Glomerulonephritis

Interstitial nephritis

Hereditary / Congenital disease

Analgesic nephropathy

Neoplasms

Less common causes

Metabolic

- Cystinosis
- Oxalosis
- Nephrocalcinosis
- Cystinuria

Vascular

- Ischemic renal disease
- Scleroderma
- Hemolytic uremic disease
- Postpartum renal failure

Autoimmune

- IgA nephropathy

Dysproteinemias

- Amyloid
- Myeloma
- Cryoglobulinemia

Hereditary

- Alport syndrome
- Fabry disease
- Tuberous sclerosis

Vasculitis

Wegeners granulomatosis

Polyarteritis nodosa

Malignancy

Renal cell carcinoma

Lymphoma

Structural

Cystic disease of kidney

Congenital and acquired abnormalities of urinary tract

PATHOGENESIS OF OCULAR FEATURES

Mild Chronic Kidney Disease patients are mostly asymptomatic although biochemical abnormalities may exist already. More severe manifestations develop as the GFR declines progressively. The following are the multisystemic complications that occur as a result of declining renal function and the accompanying ocular features.

Abnormal calcium and phosphate metabolism

There are several biochemical and hormonal abnormalities that occur in chronic renal disease that lead to abnormalities in the calcium and phosphate metabolism. Elevated levels of PTH[1] in blood and hyperplasia[1] of the parathyroid glands are seen early in the course of renal insufficiency. There is increased phosphorus retention by the failing kidney. Compensatory hyperparathyroidism results in increased phosphaturia thus maintaining serum phosphorus levels. Hyperphosphatemia becomes evident only when the GFR decreases to 20% of normal. Decreased production of 1,25- dihydroxyvitamin D in the kidney results in decreased absorption of calcium from the intestines. Persistent hypocalcemia is a powerful stimulus for the development of hyperparathyroidism. Thus hyperphosphatemia and persistant hypocalcemia leads to secondary hyperparathyroidism.

Extraskeletal calcifications are frequently encountered in patients with advanced renal insufficiency and are aggravated by persistent elevation of the

calcium – phosphate product. If the concentrations of calcium and phosphorus rise beyond a critical level, their solubility product is exceeded and precipitation occurs in tissues. Visceral deposits are an amorphous or microcrystalline compound composed of calcium, phosphate, or magnesium whereas arterial deposits consist of calcium hydroxyapatite crystals.[2] In arteries, calcium is principally deposited in the tunica media and internal elastic lamina. Thus metastatic calcification explains the deposits of calcium in cases with persistent elevated calcium.

Calcification in extraskeletal tissues can also occur due to a process known as calciphylaxis. This refers to the development of local calcification after minor local trauma in the absence of markedly elevated calcium and phosphate levels. It is a condition of induced hypersensitivity in which tissues respond to an appropriate challenger with local calcification. Renal failure acts as an indirect sensitizing calcifier, as it gives rise to increasing levels of parathyroid hormone. Minor tissue injury to the eye being the local challenger represented by devitalisation of the interpalpebral limboconjunctival and corneal epithelium being the result of markedly decreased tearflow. In these cases the calcification decreases after hemodialysis and after renal transplant.

Common ocular sites of calcium deposition include the conjunctiva (a cause of red eyes in renal patients), and Bowman's membrane (band keratopathy). These deposits tend to increase in extent in patients treated with regular dialysis and regress in patients receiving transplanted kidneys.[4]

Posterior segment calcification is less common and tends to affect the sclera and choroid. Metastatic sclerochoroidal calcifications typically occur as bilateral, multifocal, yellow fundus lesions and are usually located superotemporally.[5] Massive deposition of calcium hydroxyapatite in the previtreal space in a patient with Chronic Kidney Disease has also been reported. Retinal arteriolar calcification has also been reported in some patients.

Cardiovascular disease

Most of the patients with CKD have a higher blood pressure than the general population. Hypertension may be the primary inciting factor causing renal damage or it may be the consequence of the renal disease. Hypertensive retinopathy in varying stages is a common accompaniment in most cases of renal failure.

Anaemia

Anemia is defined as a reduction in the oxygen carrying capacity of blood, measured in the laboratory as a low hemoglobin concentratioin, or a low hematocrit (the percentage of the blood volume that is occupied by red blood cells or erythrocytes). It occurs when the balance between the normal rates of blood loss and blood production is disturbed. There are three basic mechanisms by which this occurs: (1) blood loss, (2) excessive destruction of red blood cells

(hemolysis), and (3) abnormally low production of red blood cells by the bone marrow.

In Chronic Kidney Disease, anemia is almost always present, and can be a result of any of the mechanisms listed above. However, the typical “anemia of chronic renal insufficiency” is a result of a decreased production of red blood cells by the bone marrow.

This defect in red blood cell production is largely explained by the inability of the failing kidneys to secrete the hormone erythropoietin. This hormone is a necessary stimulus for normal bone marrow to produce red blood cells. In addition, other factors associated with renal failure, including the accumulation of so-called uremic toxins, may play a role in depressing bone marrow function. Excess stores of aluminum may accumulate in the bone marrow of long term dialysis patients and can contribute to anemia as well.

Blood loss and red blood cell destruction also frequently contribute to the anemia in patients with renal failure. Platelets, which are small constituents of blood which aid in blood clotting, do not work normally in uremia. The defective blood clotting seen in uremia makes bleeding more common. Rapid bleeding causes a rapid decrease in the hematocrit and is a medical emergency. Very slow loss of blood can also cause anemia by depleting the body’s stores of iron, which the bone marrow uses to produce blood cells.

Excessive destruction of red blood cells is also seen in advanced renal failure. Normally, red blood cells survive for about four months before being destroyed. This life span is reduced in renal failure, probably because of chemical effects of uremia and decreased flexibility of the red blood cells. This hemolysis is usually mild and a person with a normal bone marrow could easily compensate for it by increasing red blood cell production. However, in renal failure, the bone marrow's capacity to compensate is diminished.

Anaemia produces a variety of ocular symptoms and signs ranging from conjunctival pallor, retinal hemorrhages, disc pallor leading to nutritional amblyopia and can also cause retrobulbar neuritis.

Malnutrition

Malnourishment is very common in Chronic Kidney Disease. It is mostly due to inadequate intake due to anorexia , acidosis and insulin resistance. There is loss of muscle weight and muscle bulk. Low levels of serum albumin, transferring and cholesterol have been observed in renal failure.

Sodium and Water balance

Sodium and water balance is not affected till very late in the course of the disease. Patients are able to maintain a good sodium and water balance until their GFR is reduced very severely (below approximately 10ml/min).

Bleeding diathesis

The main factors that contribute the bleeding diathesis in Chronic Kidney Disease are anemia, changes in vascular wall and platelet abnormalities. Reduced red cell mass or increased vessel luminal diameter (mediated by vasodilating effects of prostacyclins and nitric oxide) decreases peripheral dispersion of platelets and their contact with the vessel wall.

In uraemia, factors in the plasma prevent normal platelet adhesion and aggregation by inhibiting platelet membrane receptor – ligand interplay. Increased nitric oxide synthesis, mediated by accumulation of guanidinosuccinic acid appears to play a central causative role in disrupting normal platelet function. These factors cause overt or occult bleeding in various systems. In the eye subconjunctival hemorrhage and retinal hemorrhages occur.

Hypertension

Hypertension is frequently found in patients with primary renal disease. The pathogenesis of hypertension of chronic renal diseases generally divided into volume dependent and rennin dependent. Several mechanisms are involved in the hypertension of chronic renal disease. Renal disease causes an increase in renin, secondary hyperparathyroidism, decrease in medullipin levels, asymmetric dimethylarginine accumulation and endothelial dysfunction. These factors in turn cause an increase peripheral vascular resistance and increase in renal vascular resistance which in turn causes an increase in blood pressure.

OCULAR FEATURES OF CHRONIC KIDNEY DISEASE

Defective vision

Chronic Kidney Disease patients can manifest with defective vision due to a variety of reasons. The most common cause is cataract which may be due to the metabolic changes due to the causative factor or due to renal failure. It may also be due to the treatment modalities in the form of steroid which hasten cataract formation. The other reasons are advanced band keratopathy, secondary glaucoma, vitreous hemorrhage, retinopathy due to hypertension or diabetes [4] , optic neuropathy and infective endophthalmitis or panophthalmitis secondary to defective immunity.. Changes in the refractive status can also lead to defective vision. Most of the causes of defective vision are treatable.

Lid Edema

Lid edema[4] is a common accompaniment in patients with Chronic Kidney Disease. It may be the result of the systemic tendency to accumulate fluid in all parts of the body. This kind of edema is associated with facial puffiness. Lid edema can also occur due to the chronic irritation of the ocular surface and lids by the contents in exhaled air[13] .

Conjunctival Congestion (Red Eye)

Uremia refers to the complex and multi-organ clinical manifestations caused by accumulation of nitrogenous waste products due to renal failure. These waste products can be categorized into three main groups: 1. Small molecular weight substances eg. Urea and its subgroup 2. Middle molecular weight substances eg. beta2- Macroglobulin 3. Large molecular weight substances eg. p-cresol. Some of these uremic toxins can be exhaled through different mechanisms causing a fishy ammonia-like smell to the breath This uremic fetor is mainly attributable to the amines and more importantly, dimethylamine and trimethlamine. Increased level of some other gaseous compounds such as nitric oxide and hydrogen peroxide have also been traced in exhaled breath air of renal failure patients. All these volatile compounds have the potential ability to irritate the ocular surface and ultimately cause various ocular manifestations, especially the "red eye"[13]. The other reason could be the chronic inflammatory response to calcium accumulation in the conjunctiva[4].

Conjunctival pallor

This is due to the anaemia and malnutrition that accompany Chronic Kidney Disease.

Conjunctival degeneration

Degenerative changes in the conjunctiva is also found in patients with Chronic Kidney Disease. These changes on biopsy appear to be of elastotic type of degeneration. They are at times accompanied by deposits of calcium. Pingecula[4] along with a reduction in goblet cell density is a common finding.

Subconjunctival hemorrhage

Bleeding diathesis is found in many patients with renal failure. The multiple and complex mechanisms which are the cause of this has been discussed above. Due to the propensity for bleeding spontaneously is demonstrated by the frequent occurrence of recurrent subconjunctival hemorrhage in patients with renal failure. The degree of subconjunctival hemorrhage may vary from small innocuous spots to severe hemorrhage which involves all quadrants of the conjunctiva along with chemosis. The sclerosed conjunctival vessels secondary to hypertension may also rupture due to the fluctuating blood pressure leading to subconjunctival hemorrhage.

Dry Eye

There are a variety of factors that contribute to dryness of the eye in patients with renal failure. One of the most important factor is the reduced Goblet cell density that is associated with renal failure. The other factors act mainly by altering the ocular surface causing instability of the tear film. The

degree of dryness may vary from mild to very severe dryness leading to secondary ocular infections. The systemic causative factors like Diabetes may also play a primary role in causation of dryness.

Corneal deposits

There is a chalk like material found to be deposited on the cornea. There is a clear deposit free zone between the deposit and limbal margin. They are predominantly near the area of conjunctival degeneration. These patients show an abnormal high serum calcium levels. The clinical picture is similar to white limbus girdle of Vogt Type I. it has been found that in patients who undergo hemodialysis or those who have underwent renal transplant the deposits regress. This could be because of the fact that the secondary hyperparathyroidism starts to regress once the inciting factors are altered. If allowed to continue the deposits progress more centrally to involve a big portion of the cornea and develop into a full fledged band keratopathy.

Cataract

In patients with renal failure cataracts occurs mostly due to the inciting factor and also due to the treatment modalities. Renal failure per se does not cause cataract with increased frequency. Cataracts in renal failure is due to hypocalcemia. This requires long periods of hypocalcemia and the cataracts thus formed are of the posterior cortical variety. Cataracts due to the inciting factors like diabetes occur with increasing frequency. It is difficult to ascertain

whether the cataract is due to diabetes or due to the senile changes in patients. The other important factor that plays a significant role in cataract formation is the use of steroids and other cataractogenic medications in the treatment of renal disease. Of these cases of drug induced cataracts steroid induced cataracts are very common.

Glaucoma

Secondary glaucoma in the form of neovascular glaucoma may occur in patients with diabetes and also in hypertensives who have retinal vessel occlusion.

Retinal Detachment

The type of retinal detachment that we come across in patients with Chronic Kidney Disease is a bullous retinal detachment. Numerous hypothesis have been suggested for retinal detachments-dilutional hyponatremia, severe hypertension, ischemic infarction of choroids and choroidal spasm. Multiple factors may be operable simultaneously. The interesting part is that the retinal detachment is reversible when various causes or the complications are managed adequately and that includes renal transplantation as well. Past literatures have suggested that the occurrence or finding of retinal detachment in renal failure patients is an indication for early dialysis.

Retinopathy

Diabetic and hypertensive patients show evidence of retinopathy. If these disease processes are the main reason for the renal failure then the severity of the retinopathy is more. If they are coexistent conditions then there is not much difference in the type of retinopathy except for the occurrence of more cotton wool spots in renal patients.

Vitreous hemorrhage.

Vitreous hemorrhage is not a common accompaniment in Chronic Kidney Disease. Most of the cases of vitreous hemorrhage are due to advanced proliferative diabetic retinopathy. Untreated hypertension or accelerated hypertension in these patients can also lead to vitreous hemorrhage. The bleeding tendency in Chronic Kidney Disease patients and the anemia along with the capillary changes can also lead to vitreous hemorrhage.

Optic neuropathy

Anterior and posterior ischemic optic neuropathy can occur in these patients. It could also be secondary to diabetic papillopathy or due to accelerated hypertension.

Secondary ocular infection

Patients with renal failure are in a state of immune suppression due to defective immunity or due to chronic steroid therapy. Hence they are prone to secondary infections which can affect the ocular coats or at times may be blood borne and get seeded in the vitreous. Vision threatening infections may be uncommon but do occur and needed timely and appropriate intervention. Rarely patients have to undergo evisceration or enucleation due to the prolific infection.

PART - II

AIM OF THE STUDY

Chronic Kidney Disease affects every organ system including the eye. The requirement for a routine ophthalmic examination for all patients with chronic kidney disease and its role in the prevention of visual loss cannot be over emphasized. The aim of the study is to conduct a thorough ocular examination and to study the occurrence of various ocular manifestations exhibited by patients with Chronic Kidney Disease and to analyse the findings.

MATERIALS AND METHODS

This is a Cross Sectional, Descriptive, Non interventional, Hospital based study. The period of study was for 15 months, from August 2010 to October 2011.

Institutional Ethical Committee approval for conducting the study was obtained.

Patients presenting to Department of Nephrology, Stanley Medical College diagnosed with Chronic Kidney Disease were examined for ocular manifestations at the Department of Ophthalmology, Stanley Medical College. Sampling technique was consecutive and 100 patients (200 eyes) were enrolled in this study.

Importance of ocular evaluation were explained to the patients. Evaluation procedures were explained and an informed consent was obtained. After obtaining consent 200 eyes of the enrolled patients were examined thoroughly.

Results of blood and urine investigations performed at Nephrology department were collected.

The following ocular evaluation were conducted.

1. Relevant ocular history
2. Best corrected visual acuity
3. Detailed slit lamp examination of anterior segment
4. Posterior segment evaluated with indirect ophthalmoscope and Slit lamp biomicroscopy using 90D. Hypertensive retinopathy if present was graded using Keith, Wagner and Barker classification and diabetic retinopathy was graded using ETDRS system.
5. Schirmer's test using Whatman filter paper strip.
6. Intraocular pressure was measured with Goldmann applanation tonometer.
7. Visual field analysis using Octopus perimeter, B scan ultrasonograghy and optical coherence tomography was done whereever indicated

.

The results thus obtained were tabulated and analysed. Statistical analysis was done using the Chi square test

Inclusion criteria:

1. All stages of Chronic kidney disease .

2. Renal transplant recipients.

3. Duration of renal disease for more than 3 months.

4. Age group between 20 years to 70 years.

Exclusion criteria:

1. Cases with renal disease of unknown etiology

2. Cases with acute fulminant disease

3. Cases with known pre-existing ocular disease

OBSERVATION AND DISCUSSION

In this study, a total of one hundred cases of Chronic Kidney Disease who were referred from the Department of Nephrology, Stanley Medical College, Chennai to the Department of Ophthalmology were enrolled in the study after obtaining informed consent and were subjected for thorough ocular examination .

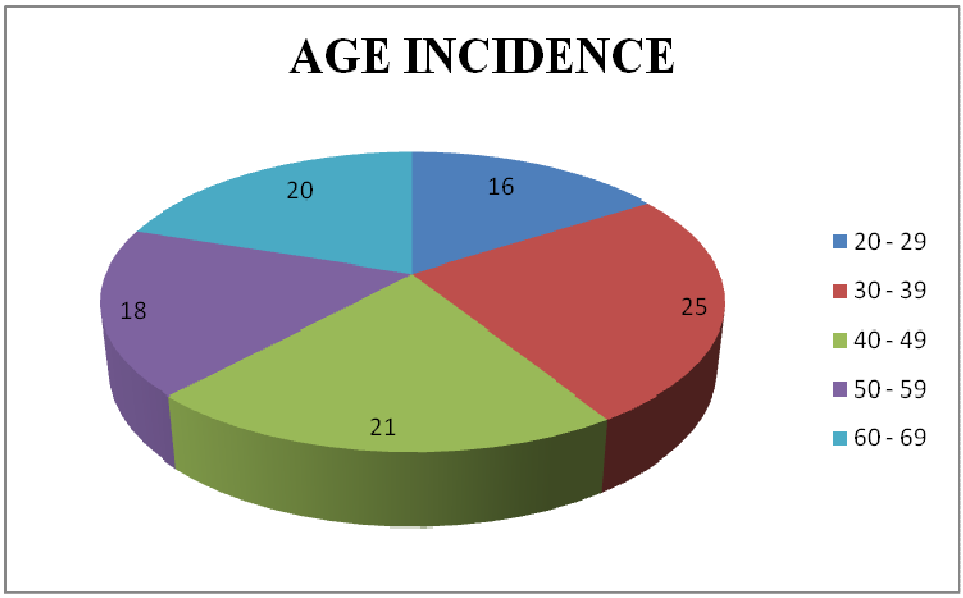
AGE INCIDENCE

The Age groups of the patients included in the study were as follows.

Table. 1 : Age Incidence

Age Group	No. of Cases	%
20 – 29	16	16
30 – 39	25	25
40 – 49	21	21
50 – 59	18	18
60 – 69	20	20

The age distribution in the study group was more or less even with the patients in the age group 30 – 39 slightly more than the rest of the cohort.

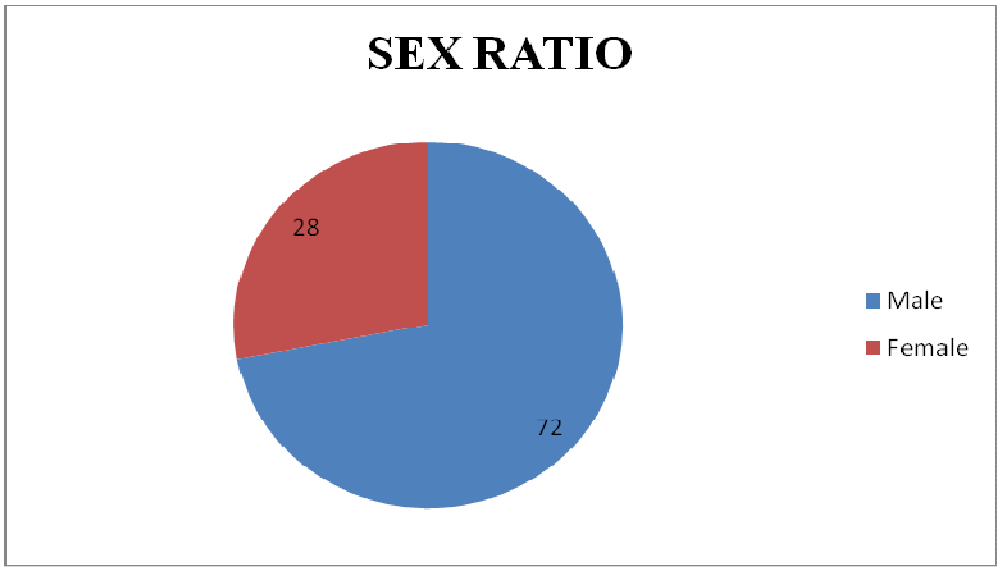


SEX RATIO

Male patients formed 72% of the total patients in the study group. The average male : female ratio was 2.6:1. The gender difference was more in the age groups 30- 39 and 40 – 49 which showed a ratio of 4:1 and 4.25:1 respectively. The ratio was almost equal in the age group 50 – 59 which showed a ratio of 1.25:1.

Table. 2 : Sex Ratio

Sex	No. Of Cases	%
Males	72	72
Females	28	28

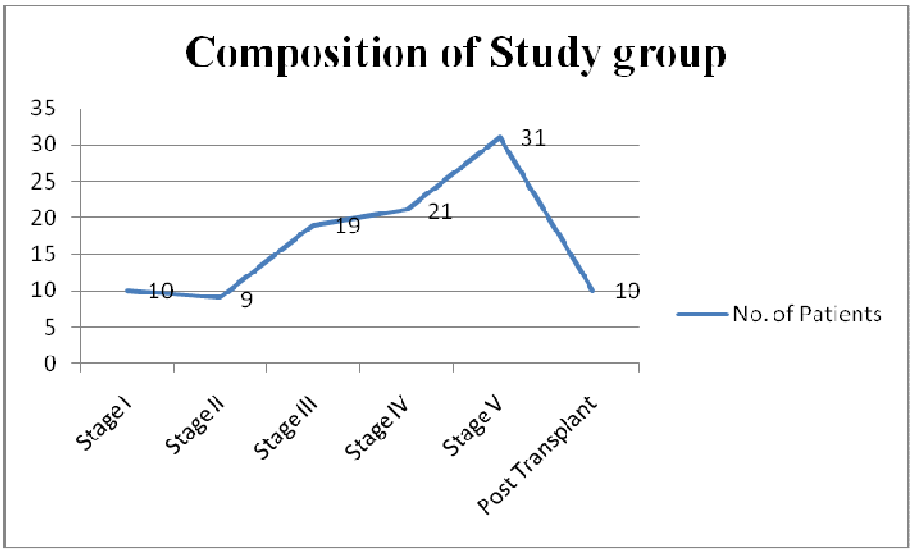


STAGES OF CKD IN STUDY POPULATION

The study group consisted of 31 patients with Stage V disease, 21 with Stage IV and 19 patients with Stage III disease. 29 patients were evenly distributed in groups of stage I,II and post transplant category.

Table. 3 : Stages Of CKD In Study Population

Stage of CKD	No. of Patients	%
Stage I	10	10
Stage II	9	9
Stage III	19	19
Stage IV	21	21
Stage V	31	31
Post Transplant	10	10



The Study of Ocular evaluation in patients with chronic renal failure published in Nepal Medical College Journal in 2008 by L.Bajracharya et al[4] showed an almost equal distribution of study population in the various stages of chronic kidney disease. But in this study , patients were predominantly placed in the advanced stages of the disease.

50.9% of patients had stage V disease and 23.67% had stage IV disease as per 5 year Cumulative report of Chronic Kidney Disease Registry of India[25] 2010

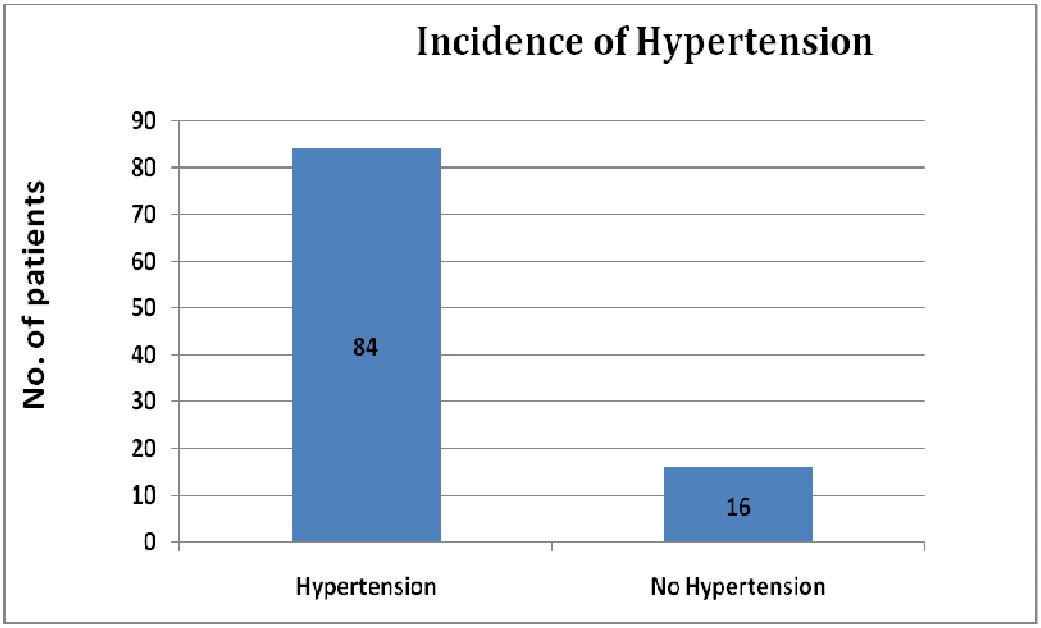
Post renal transplant patients were not included in both of the above mentioned studies.

INCIDENCE OF HYPERTENSION

The study group consisted predominantly of hypertensive patients. They constituted 84% of the patients. Of the 100 patients who were studied 34 were both hypertensive and diabetic.

Table 4. Incidence of Hypertension

Disease	No. of patients	%
Hypertensives	84	84
Non Hypertensives	16	16



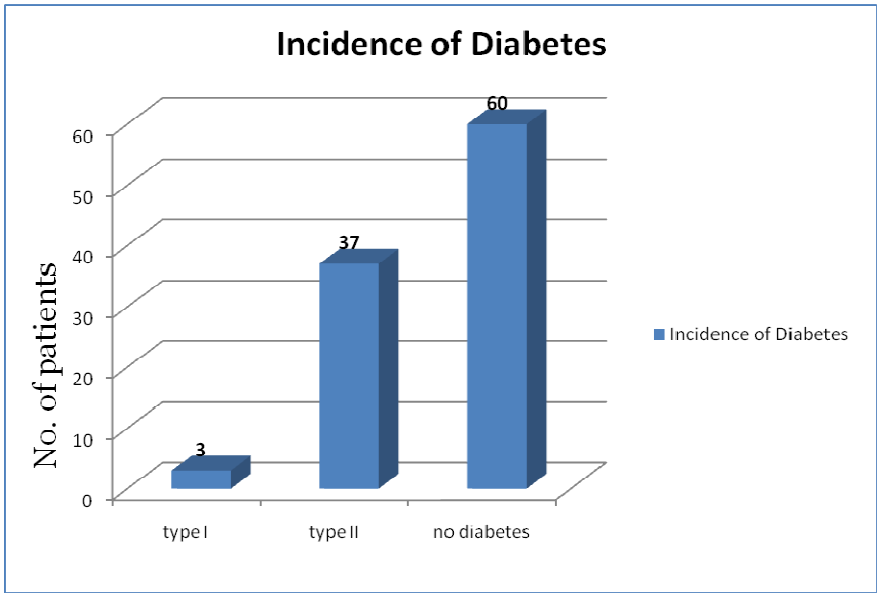
74.7% of patients were hypertensives in the 5 year Cumulative report of CKD Registry of India[25] 2010, which is comparable to this study.

INCIDENCE OF DIABETES

Diabetic patients constituted about 40% of the study group out of which 37% were suffering from type II diabetes and only 3% were suffering from type I Diabetes. 60% of the patients were nondiabetics. 17 diabetic patients were on Inj.Insulin and the rest were on oral hypoglycemic agents.

Table. 5 : Incidence of Diabetes

	No. of Patients	%
Type I	3	3
Type II	37	37
Non Diabetics	60	60



In the 5 year Cumulative report of Chronic Kidney Disease Registry of India[25] 2010, 38.4% of the study group had diabetes of which 9.9% belonged to type I DM and 90.1% belonged to type II group. This is similar to this study were type I diabetics constituted 7.5% of the diabetic population and type II constituted 92.5%.

CAUSE OF CHRONIC KIDNEY DISEASE

In this study, out of the 100 patients , 90 patients had chronic kidney disease and 10 patients were renal transplant recipients.

Table 6. Cause Of Chronic Kidney Disease

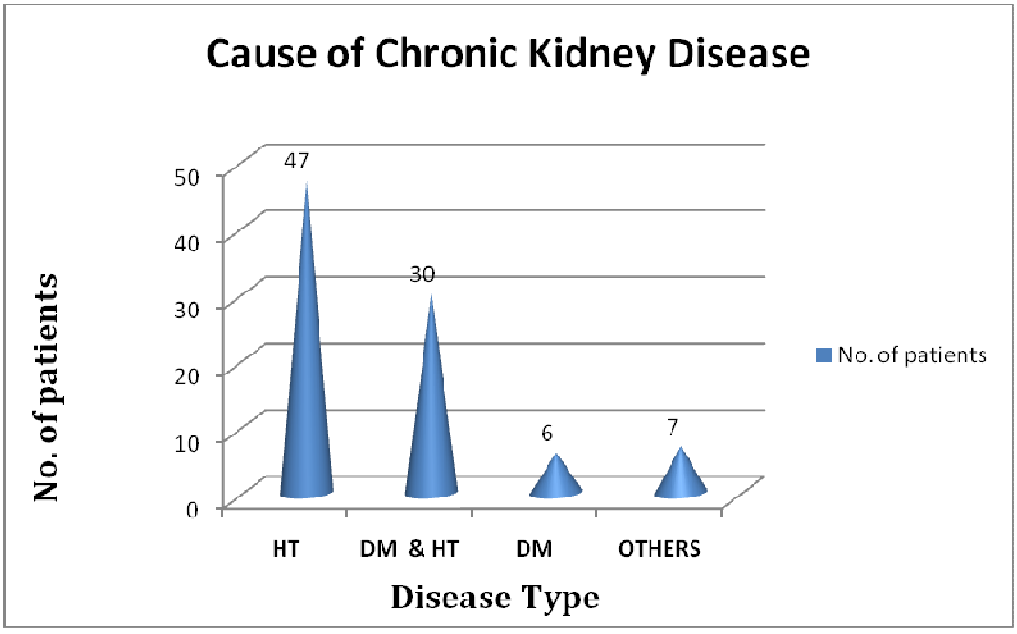
Disease	No. of patients	%
HT	47	52.2
DM & HT	30	33.3
DM	6	6.7
Others	7	7.8
TOTAL	90	100

Out of the 90 patients suffering from CKD 6 were having only diabetes and 47 were hypertensives. 30 patients had both diabetes and hypertension and

7 patients had renal disease due to other causes like Glomerulonephritis, Analgesic nephropathy , IgA nephropathy etc.

Of the 10 post renal transplant patients, 3 had hypertension, 4 had diabetes and hypertension.

Hypertension was the single main cause of Chronic Kidney Disease in this study contributing to 52.2%. 33.3% of patients had both diabetes and hypertension.



In the Study of Ocular evaluation in patients with chronic renal failure published in Nepal Medical College Journal in 2008 by L.Bajracharya et al[4] the commonest cause of Chronic Kidney disease was hypertension(36.1%).

In the 5 year Cumulative report of Chronic Kidney Disease Registry of India[25] 2010, Diabetic nephropathy was the most common cause of Chronic

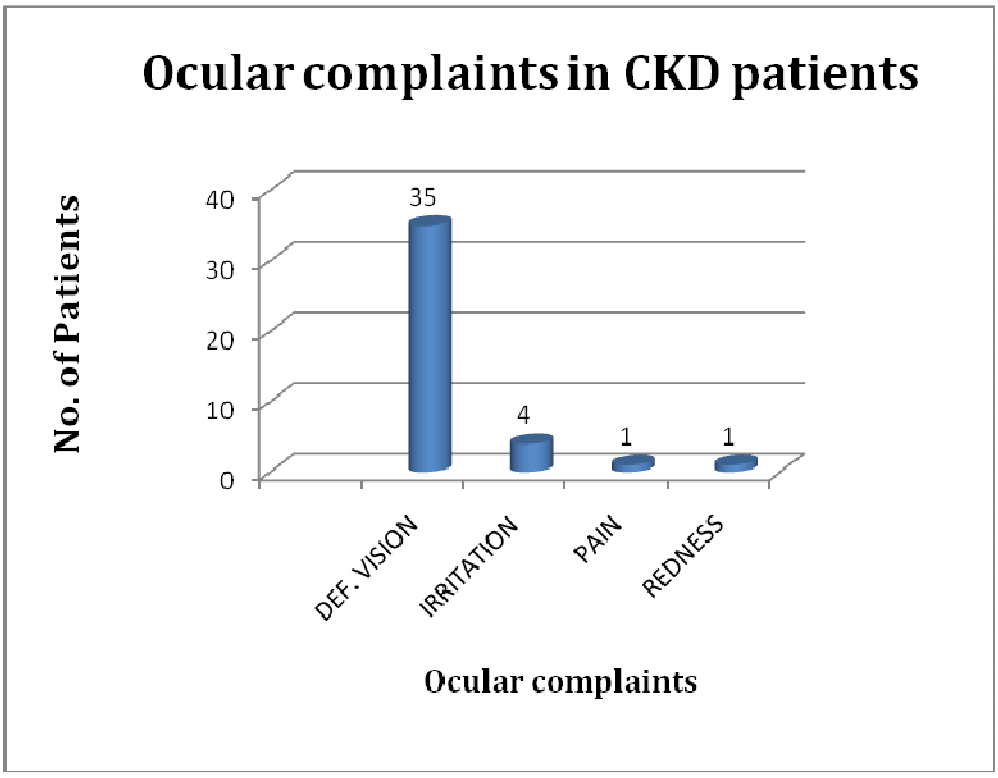
Kidney Disease contributing to 31.2%. Hypertensive nephrosclerosis constituted 12.18% .

OCULAR SYMPTOMS IN PATIENTS WITH CKD

With regards to the ocular complaints in patients screened, 59% of patients had no ocular complaints and only 41% complained of some form of ocular discomfort . 85.4 % of patients with ocular complaints , had defective vision, 9.8% of patients had ocular irritation ,2.4% of patients had pain and discharge each .

Table 7. Ocular Symptoms in Patients with CKD

TYPE OF COMPLAINT	NO. OF EYES	%
Defective Vision	35	85.4
Irritation	4	9.8
Pain	1	2.4
Redness	1	2.4
Total	41	100



Most of the patients with ocular complaints were having their eyes checked for the first time which showed the lack of awareness about the potential ocular complications in CKD.

In the study done by Easterbrook et al, published in the British journal of Ophthalmology (1970), all patients had excellent visual acuity. Whereas L.Bajracharya's[4] study of ocular evaluation in patients with chronic renal failure published in Nepal Medical College Journal in 2008 claimed that 62% of patients had defective vision and 29% had ocular irritation.

BEST CORRECTED VISUAL ACUITY

Best corrected visual acuity was tabulated .71.5% of the total patients enrolled were with vision 6/18 or better .In this study, according to WHO criteria ,18.5% were visually impaired (6/24 – 6/60) and 10% were in the category of legally blind (vision <6/60).

This finding is comparable to the Study of Ocular evaluation in patients with chronic renal failure published in Nepal Medical College Journal in 2008 by L.Bajracharya et al[4] which showed that 76.6% of patients had good vision and patients with impaired vision were 11.7% and those who were legally blind were 11.7%.

Table. 8 : BEST CORRECTED VISUAL ACUITY

BCVA	Stage I	Stage II	Stage III	Stage IV	Stage V	Post Transplant	Total eyes	%
>/= 6/18	14	11	30	30	40	18	143	71.5
6/24 - 6/60	2	3	6	12	12	2	37	18.5
< 6/60	4	4	2	0	10	0	20	10
Total							200	100

Patients with visual acuity less than 6/60 were significant with

p value<0.05

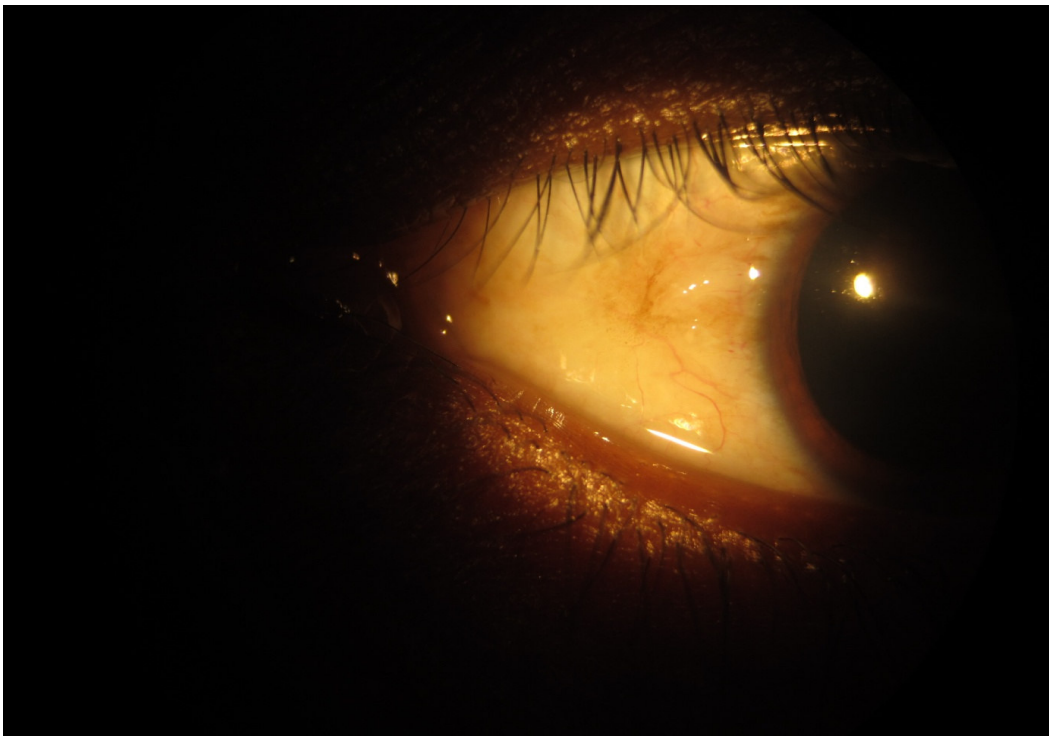
ANTERIOR SEGMENT FINDINGS IN DIFFERENT STAGES OF CHRONIC KIDNEY DISEASE

Of the 200 eyes included in the study 160 eyes showed changes in the Anterior segment . 57.5% of eyes had cataract , 16.9% of eyes showed lid edema and 17.5% of eyes had conjunctival pallor. The other ocular findings that were noticed were iridocyclitis, defective extraocular movements and pingecula.

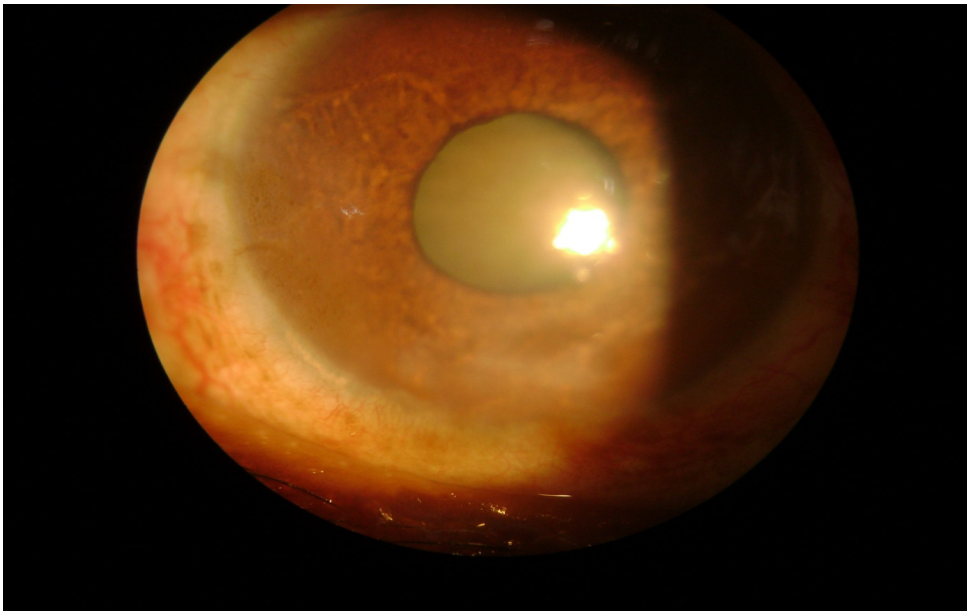
Table. 9 : Anterior Segment Findings in Different Stages of CKD

	Stage I	**Stage II**	**Stage III**	**Stage IV**	**Stage V**	**Post Transplant**	**Total eyes**	**%**
Lid edema	4	2	4	8	9	0	27	16.9
Conj. Pallor	6	2	8	4	8	0	28	17.5
Pingecula	2	0	0	2	4	0	8	5.0
Cataract	5	2	10	31	38	6	92	57.5
EOM Restriction	0	0	0	0	1	0	1	0.6
Proptosis	0	0	0	0	1	0	1	0.6
Iridocyclitis	0	0	0	0	0	1	1	0.6
Band shaped Keratopathy	0	0	1	1	0	0	2	1.3
Total	17	6	23	46	61	7	**160**	100.0

PINGECULA



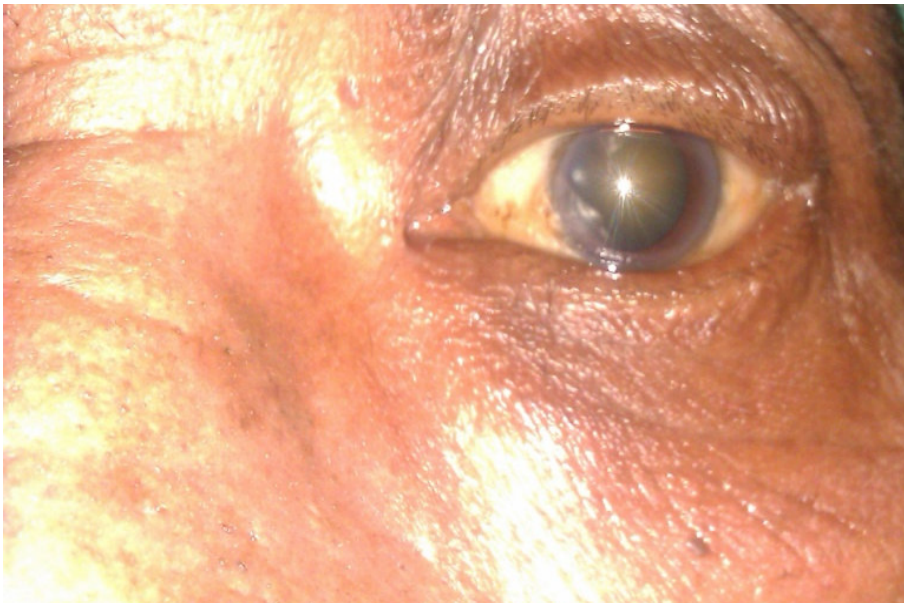
BAND SHAPED KERATOPATHY

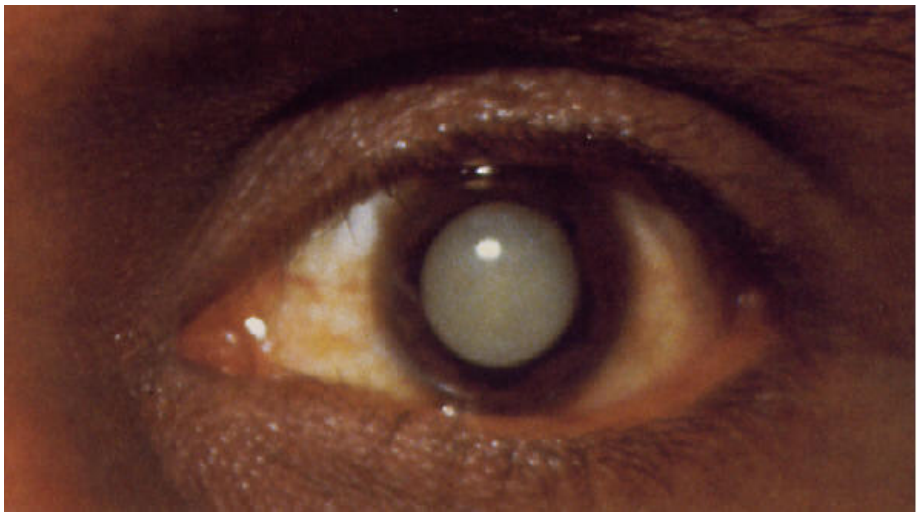


CONJUNCTIVAL PALLOR

LID EDEMA

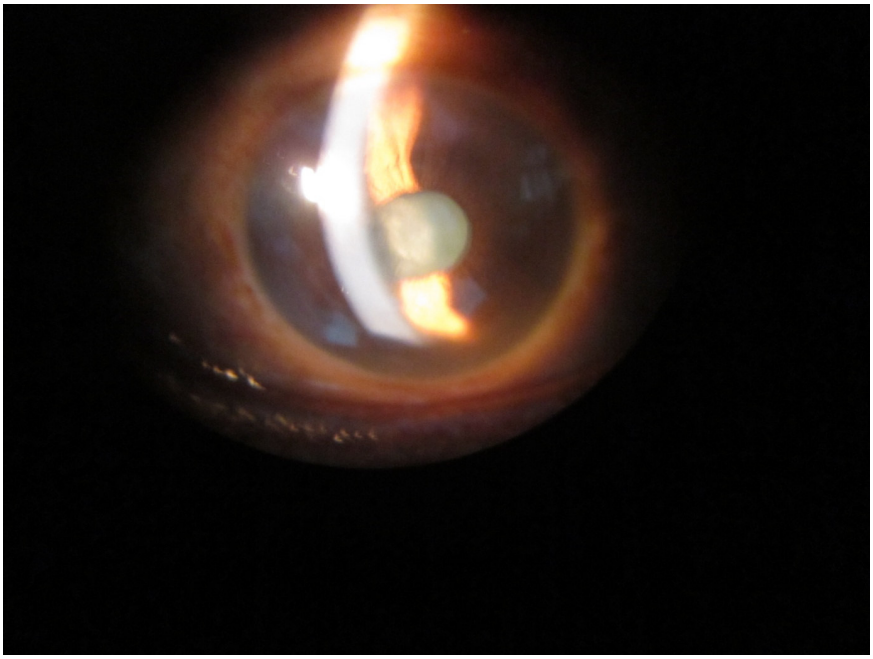
CATARACT





PLASTIC IRIDOCYCLITIS IN A POSTRENAL TRANSPLANT

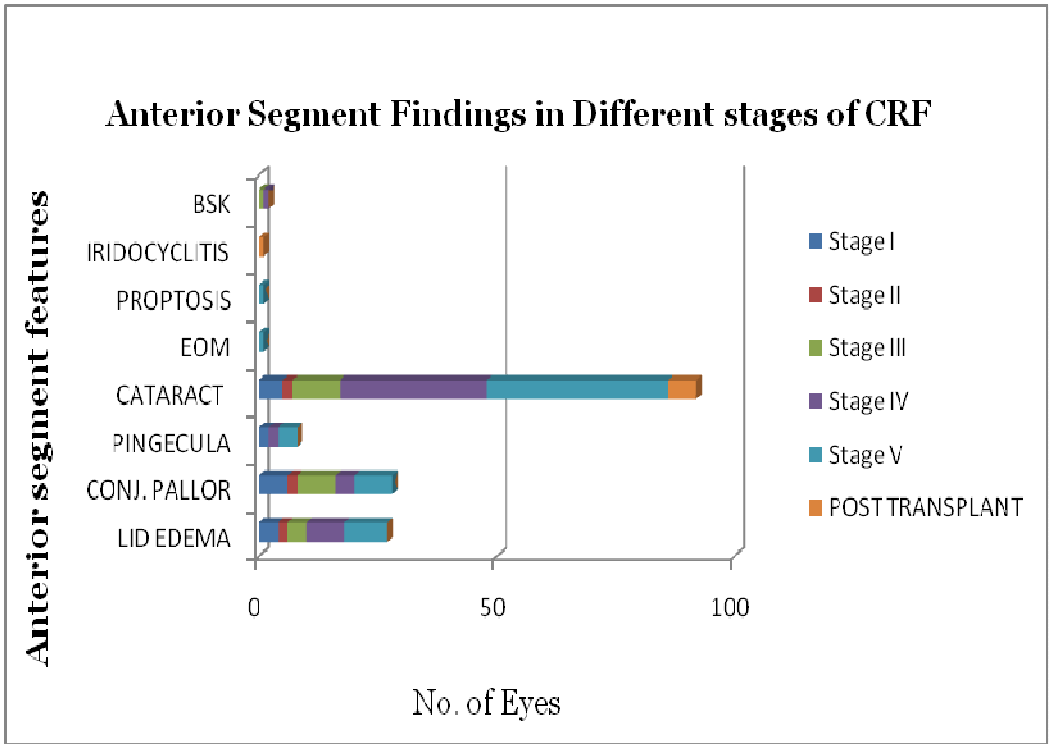
PATIENT ON IMMUNOSUPPRESION



DIABETIC PATIENT WITH PROPTOSIS AND EOM RESTRICTION.

When the incidence of these symptoms in different stages of Chronic Kidney Disease were studied it was found that cataract was found in 38 eyes of patients with end stage or Stage V renal disease. Stage V group patients also exhibited the most number of anterior segment signs (61 eyes). In patients with stage IV and Stage III renal disease 46 and 23 eyes showed signs.

On analysing the findings ,the significant findings in the anterior segment were cataract ($p < 0.02$) and conjunctival pallor($p < 0.02$) .



One patient , who was a diabetic with stage V CKD ,had proptosis with extraocular movement restriction in the left eye. Investigations revealed

mucormycosis of the maxillary sinus with orbital involvement. The patient underwent surgical debridement of the sinus with appropriate medical therapy.

Plastic iridocyclitis was found in a renal transplant patient with transplant dysfunction. The patient was on steroids and other immunosupressive agents.

CATARACT IN PRESENILE PATIENTS

An observation of the incidence of cataract in the presenile age group (<=50) was done. This showed that of the 65 patients (130 eyes) , 53eyes had some form of cataract and 11 eyes have undergone cataract extraction with a posterior chamber intraocular lens in place.

Table. 10 : Cataract in Presenile Patients

	No. of eyes	**%**
Cataractous eyes	53	40.8
PCIOL	11	8.5
TOTAL	64	49.3

Thus 49.3% of presenile patients had cataract which is significant.

INCIDENCE OF OCULAR SURFACE DISEASE IN CKD

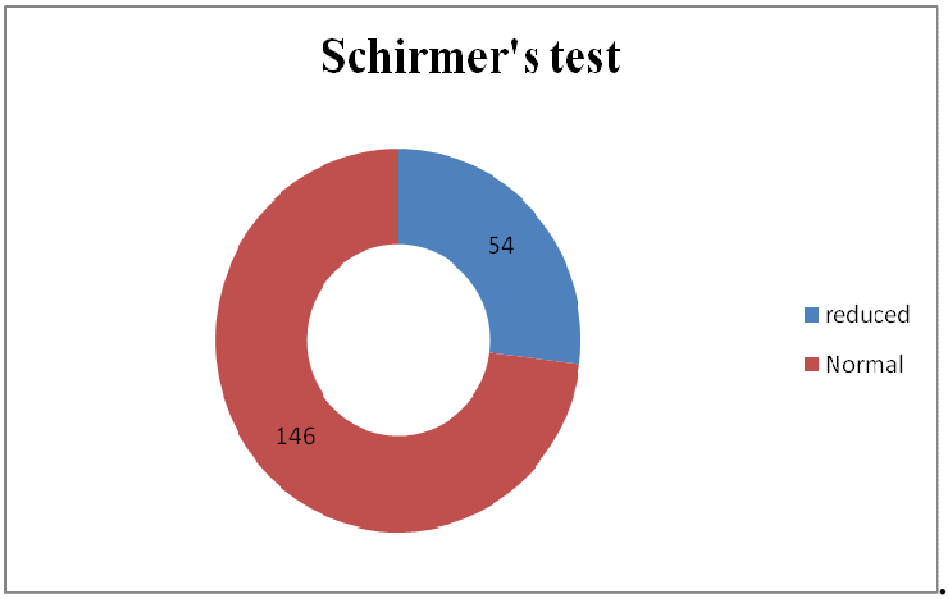
A reduced Schirmer's value was noted in 54 eyes and was normal in 146 eyes. The incidence of ocular surface disease in the study was 27%.

Table. 11 : Incidence Of Ocular Surface Disease in CKD

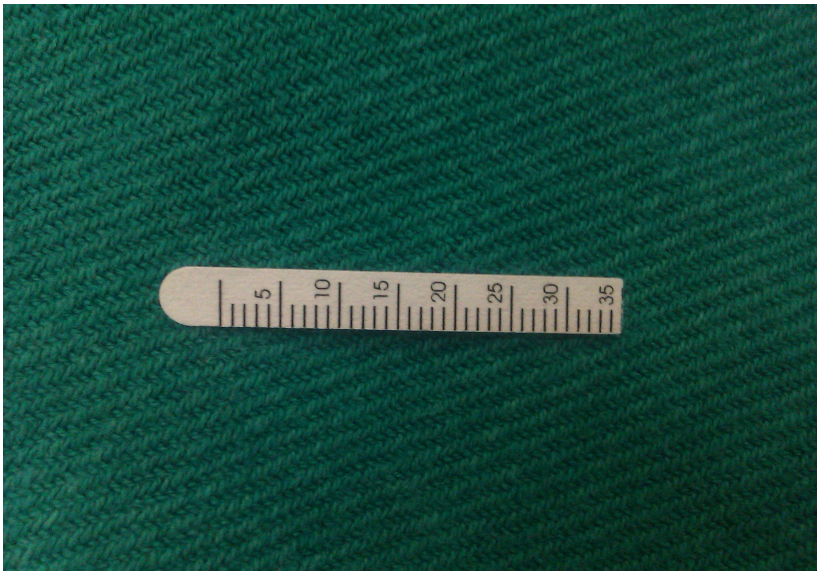
Schirmer's test	No. of eyes	%
Reduced	54	27
Normal	146	73

The presence of ocular surface disease in this study was significant with p value <0.01.

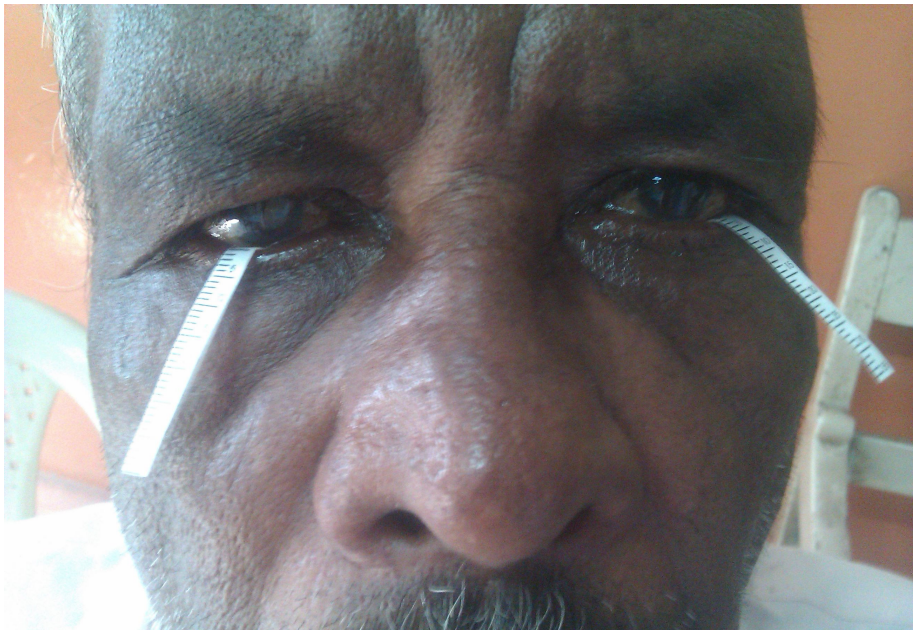
In the Study of Ocular evaluation in patients with chronic renal failure published in Nepal Medical College Journal in 2008 by L.Bajracharya et al[4] the presence of dry eye was not a significant finding as only 7.5 % of the Chronic Kidney Disease patients had dry eye as compared to 27% in this study.


Schirmer's test
54
146
reduced
Normal

WHATMAN FILTER PAPER STRIP



SCHIRMER'S TEST



INCIDENCE OF HYPERTENSIVE RETINOPATHY IN CKD

In this study there was a total of 84 patients (168 eyes) with hypertension, of which 92 eyes (46%) showed hypertensive changes.

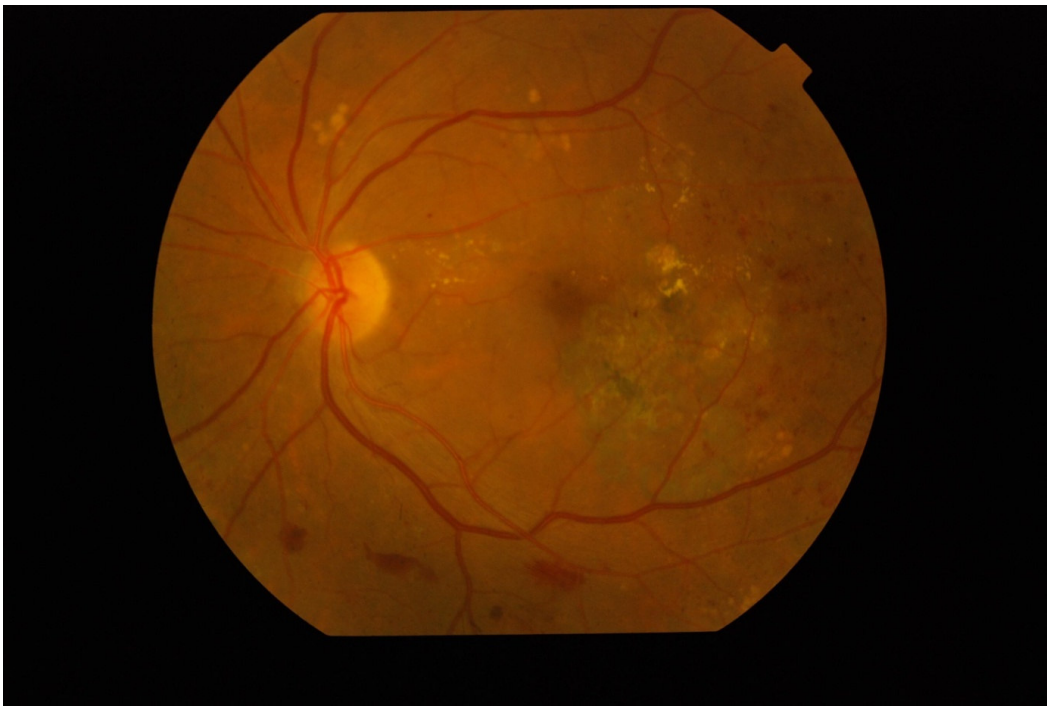
43 eyes had Grade III retinopathy making it the most common hypertensive retinopathy. Higher grades of hypertensive retinopathy was found in advanced stages of CKD i.e 24 eyes in stage IV and 23 eyes in stage V.

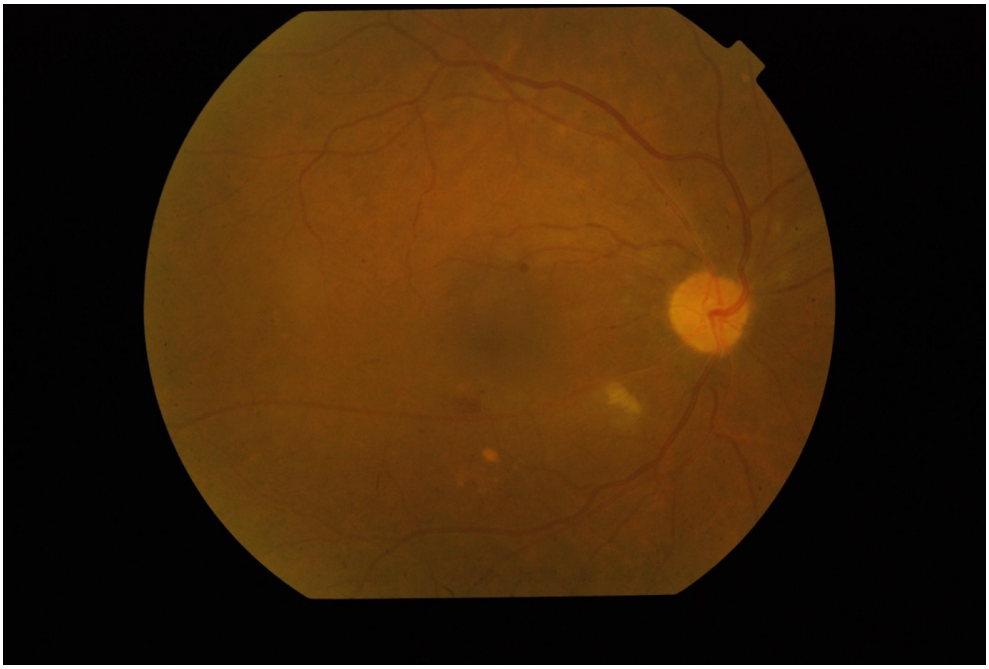
92 eyes (46%) out of the 200 under study showed hypertensive retinopathy In the study by L.Bajracharya et al[4] 48% of total patients had hypertensive retinopathy and retinopathy was more common in patients in stage IV and Stage V disease which is comparable to this study.

Table. 12 : Incidence of hypertensive retinopathy in CKD

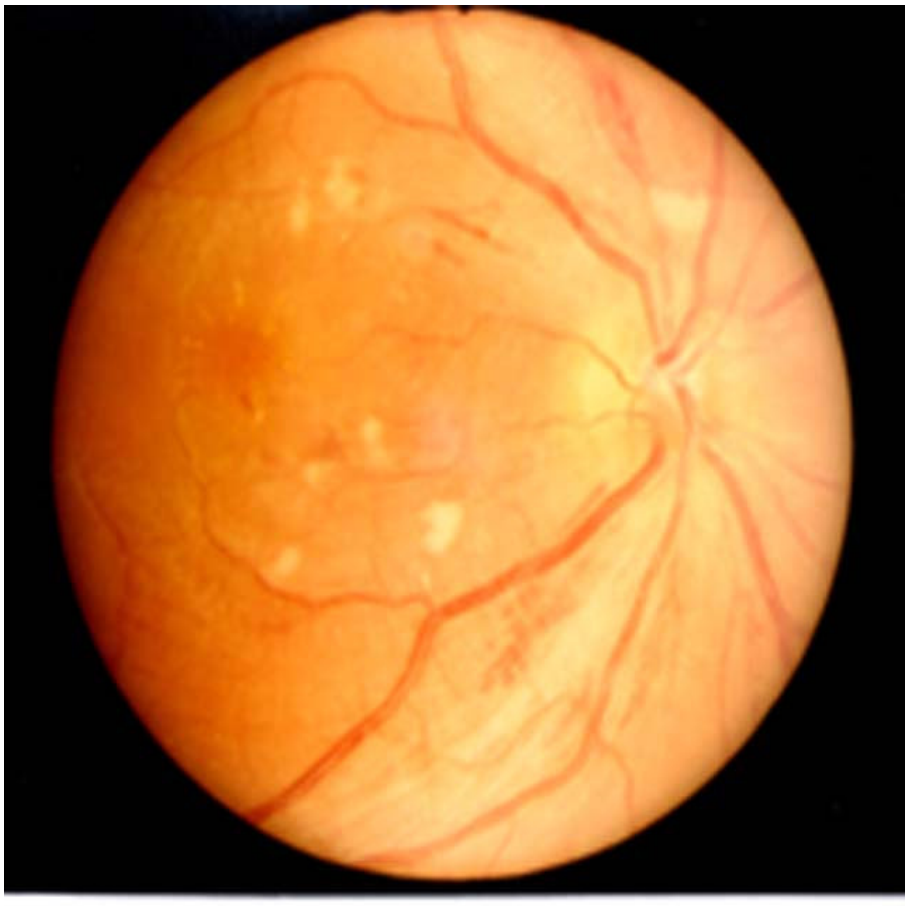
	Stage I	**Stage II**	**Stage III**	**Stage IV**	**Stage V**	**Post Transplant**	**Total eyes**
Grade I HR	0	2	5	2	5	3	17
Grade II HR	5	2	9	3	8	1	28
Grade III HR	5	4	5	19	8	2	43
Grade IV HR	0	2	0	0	2	0	4
Total	10	10	19	24	23	6	**92**

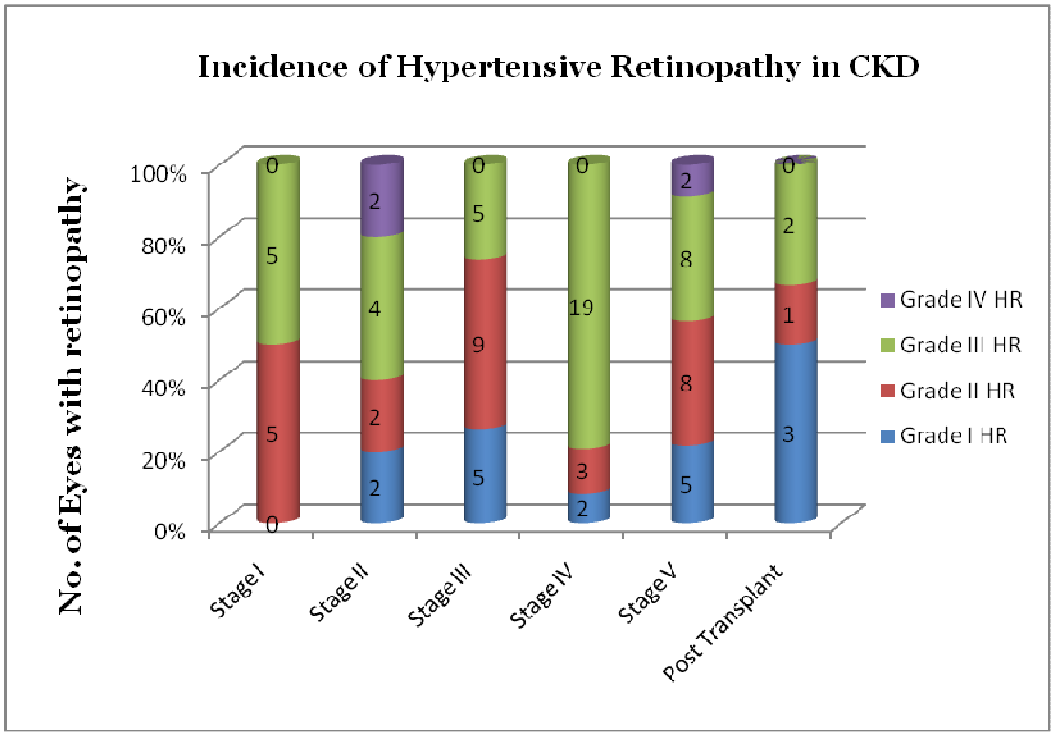
GRADE III HT RETINOPATHY





GRADE IV HYPERTENSIVE RETINOPATHY





INCIDENCE OF DIABETIC RETINOPATHY IN CKD

Of the 40 patients (80eyes) with diabetes , 51 eyes i.e., 63.75% exhibited some degree of diabetic retinopathy.

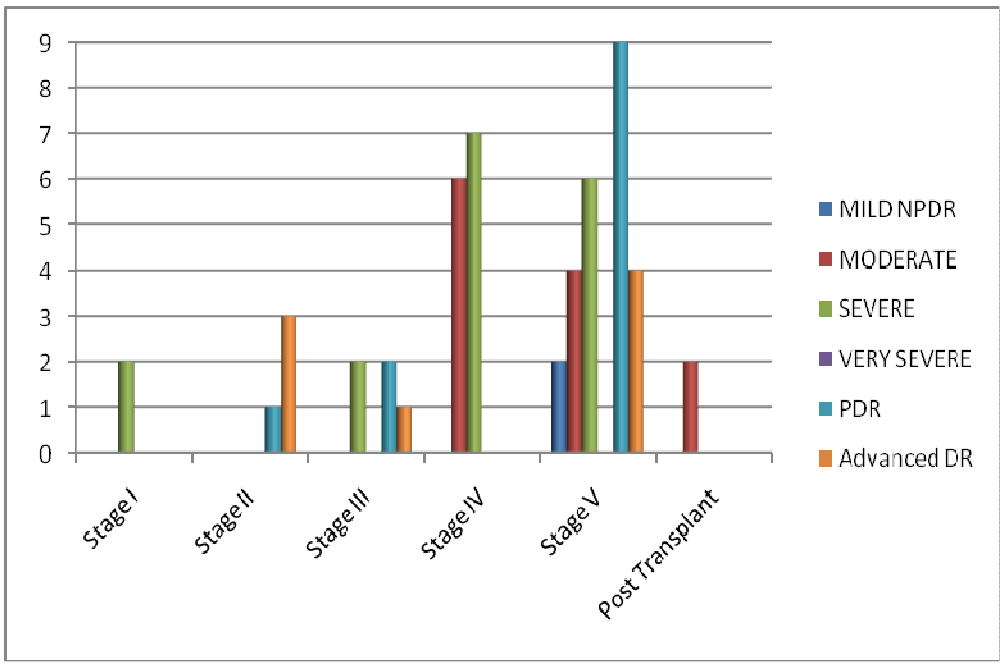
Diabetic retinopathy was most common in patients with Stage V renal disease with 25 eyes out of the 51 eyes belonging to this group followed by stage IV having 13 eyes with diabetic retinopathy.

8 eyes had advanced diabetic eye disease of which 4 eyes belonged to stage V. Proliferative diabetic retinopathy was also more common in stage V

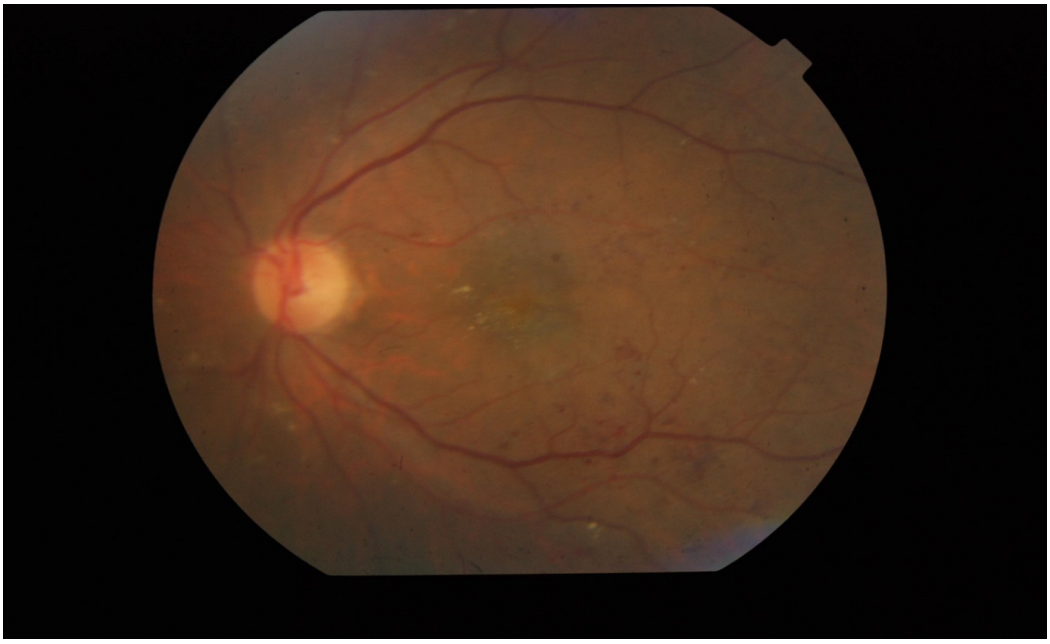
CKD similar to what was observed in the WESDR study where prevalence of PDR was much higher in patients with persistent microalbuminuria.

Severe NPDR was the most common stage of diabetic retinopathy which was found in 17 eyes. Second most common diabetic retinopathy was moderate NPDR and PDR having 12 eyes each.

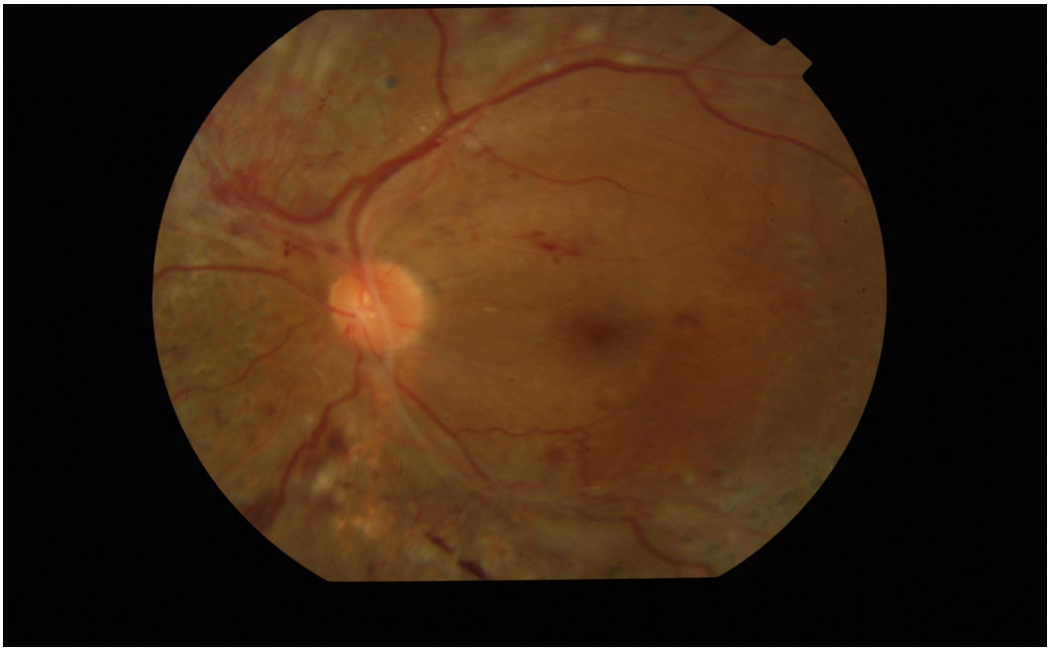
Clinically significant macular edema was present in 6 eyes in various grades of Diabetic retinopathy. All of them belonged to stage V CKD. In the Wisconsin Epidemiologic study of Diabetic Retinopathy (WESDR)[27,28] the presence of gross proteinuria suggestive of CKD was associated with increased risk of developing Diabetic Macular Edema so also in this study most of the patients with Clinically significant macular edema belonged to advanced kidney disease group.



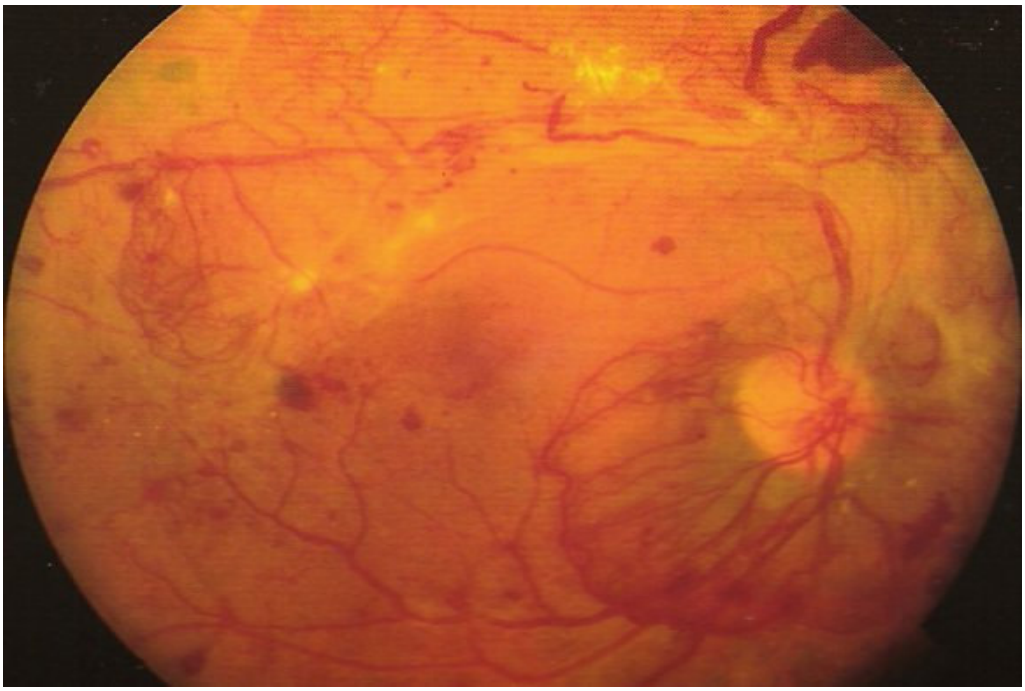
SEVERE NPDR WITH CLINICALLY SIGNIFICANT MACULAR EDEMA



PROLIFERATIVE DIABETIC RETINOPATHY



ADVANCED DIABETIC EYE DISEASE



PDR WITH VITREOUS HEMORRHAGE

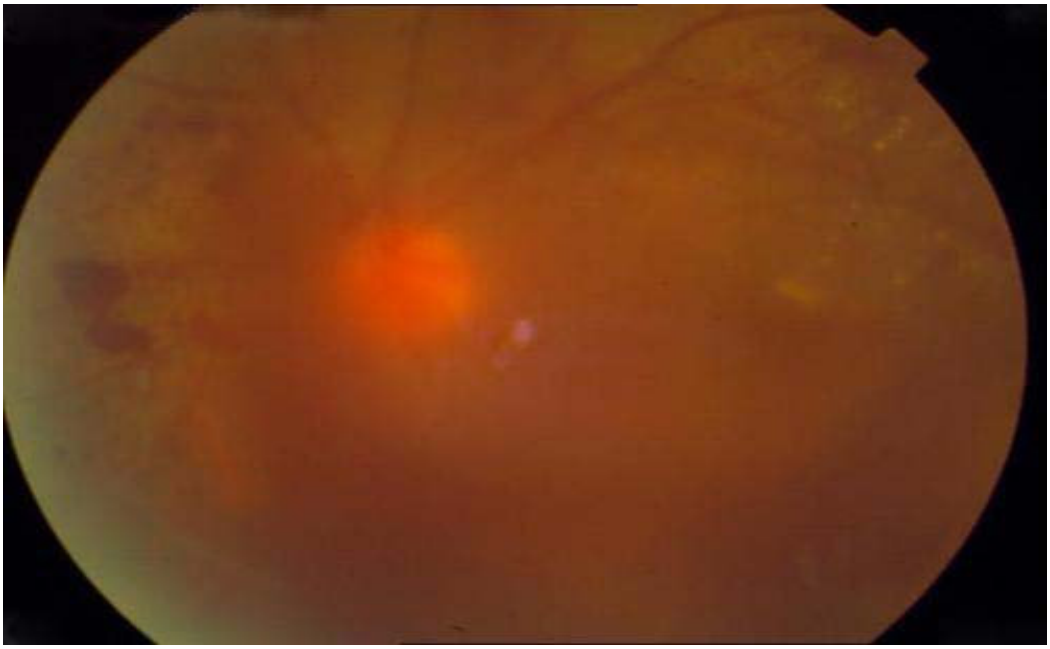


Table. 13 : Incidence of Diabetic Retinopathy In CKD

	Stage I	Stage II	Stage III	Stage IV	Stage V	Post Transplant	Total eyes
Mild NPDR	0	0	0	0	2	0	2
Moderate NPDR	0	0	0	6	4	2	12
Severe NPDR	2	0	2	7	6	0	17
Very Severe NPDR	0	0	0	0	0	0	0
PDR	0	1	2	0	9	0	12
Advanced DR	0	3	1	0	4	0	8
Total	2	4	5	13	25	2	**51**

L.Bajracharya et al[4] demonstrated that 88.3% of diabetics had retinopathy whereas in this study 63.75% of diabetics had retinopathy. Both the studies were similar in the fact that more number of patients in stage V showed Diabetic retinopathy.

In the Andhra Pradesh Eye Diseases study(APEDS)[28] the prevalance of diabetic retinopathy was 22.4%. In the Chennai Urban Rural Epidemiology Study (CURES)[28] the overall prevalence of diabetic retinopathy among the sample of diabetic patients was 17.6% .

In this study group of CKD patients with diabetes , the patients with diabetic retinopathy is 63.75% which is significantly more when compared to diabetic retinopathy prevalence in diabetic population without CKD.

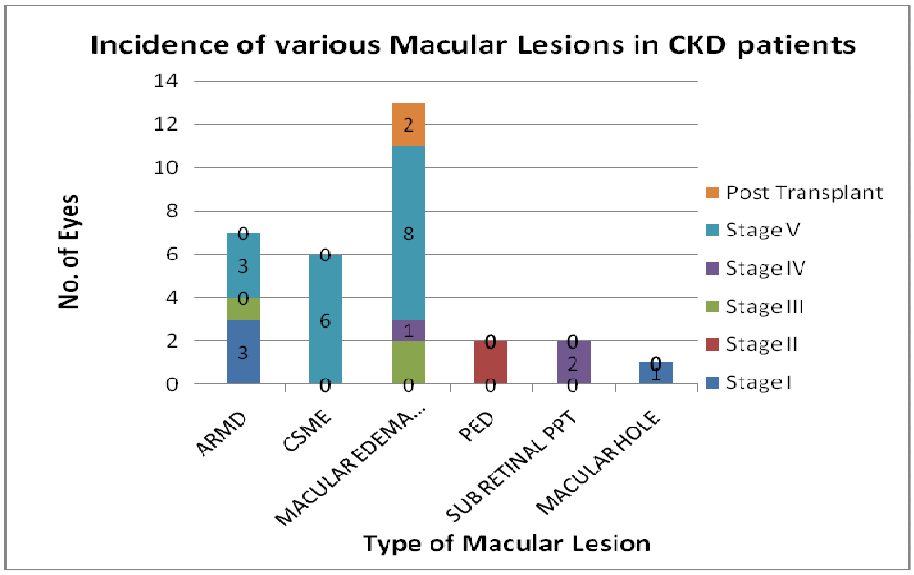
MACULAR FINDINGS IN STUDY GROUP

31 eyes from the study group showed changes in the macula. The most common sign was macular edema which was found in 13 eyes. This group had a mixed population of diabetic and hypertensive patients. Other common findings in the macula were age related macular degeneration changes and clinically significant macular edema in diabetic patients. PED, subretinal precipitates and macular hole .

Macular edema was more common in stage V CKD.

Table. 14 : Macular Findings

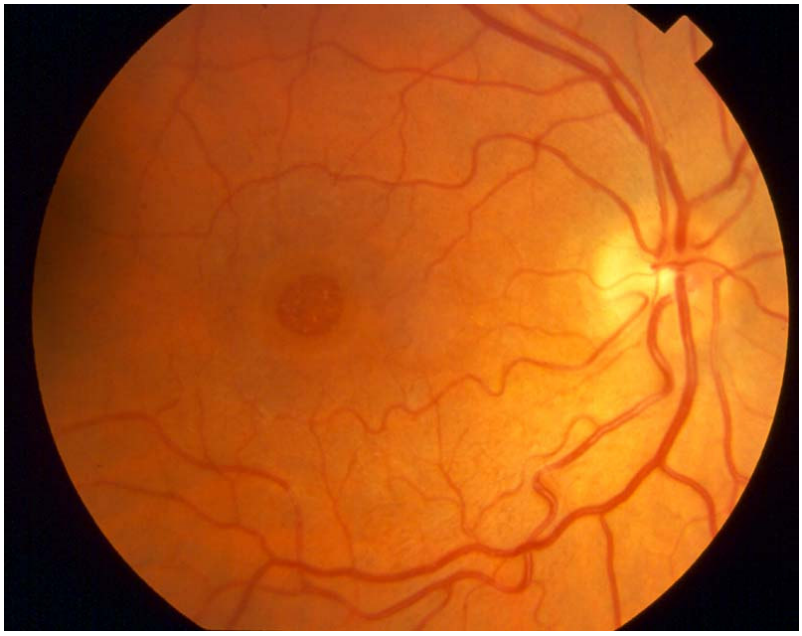
	Stage I	Stage II	Stage III	Stage IV	Stage V	Post Transplant	TOTAL EYES
ARMD	3	0	1	0	3	0	7
CSME	0	0	0	0	6	0	6
Macular Edema (DM & HT)	0	0	2	1	8	2	13
PED	0	2	0	0	0	0	2
Sub Retinal PPT	0	0	0	2	0	0	2
Macular Hole	1	0	0	0	0	0	1
Total							31

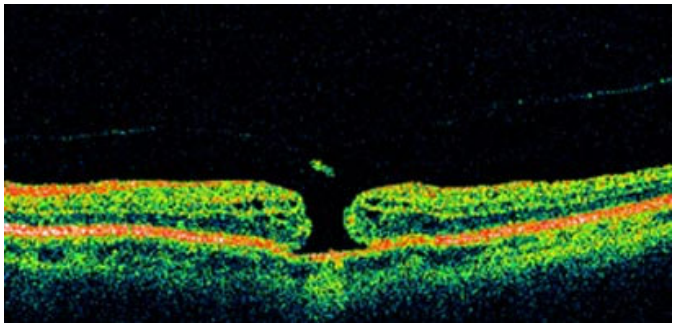

Incidence of various Macular Lesions in CKD patients
No. of Eyes
Type of Macular Lesion
ARMD
CSME
MACULAR EDEMA...
PED
SUB RETINAL PPT
MACULAR HOLE
Post Transplant
Stage V
Stage IV
Stage III
Stage II
Stage I

MACULAR EDEMA

CSME

MACULAR HOLE





OTHER POSTERIOR SEGMENT FINDINGS

When diabetic and hypertensive retinopathies as well as macular symptoms were excluded there were some posterior segment findings exhibited by 11 eyes from the study group. 4 eyes had a raised CD ratio. BRVO was found in 2 eyes of 2 patients. 3 eyes had disc pallor all of them belonged to stage IV.

Table. 15 : Other Posterior Segment Findings

	Stage I	Stage II	Stage III	Stage IV	Stage V	Post Transplant	Total
BRVO	0	0	0	2	0	0	2
CRAO	0	0	0	0	2	0	2
RAISED CD RATIO	2	0	2	0	0	0	4
Disc Pallor	0	0	0	3	0	0	3

Two patients had raised CD ratio. IOP and fields were normal in both the patients. B scan was done in patients with m edia opacities did not show any positive findings.

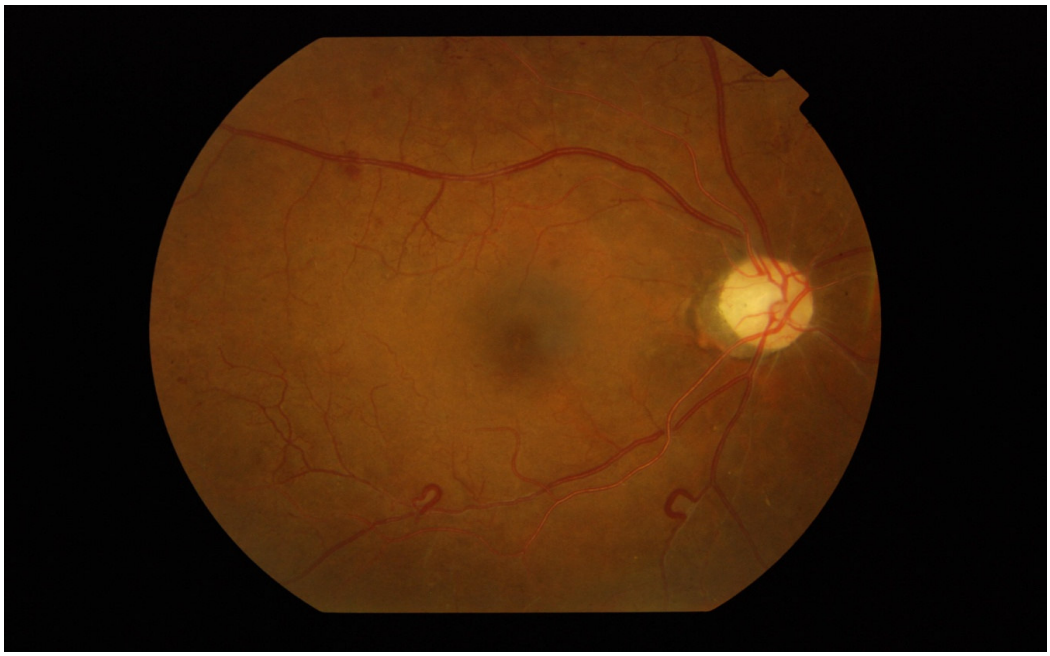
Old CRAO was found bilaterally in one patient. The patient was a hypertensive patient and had grade III hypertensive retinopathy along with findings of old CRAO.

BRANCH RETINAL VEIN OCCLUSION

DISC PALLOR

FUNDUS PICTURE OF A PATIENT WITH BE : OLD CRAO WITH GRADE III HT RETINOPATHY

RE



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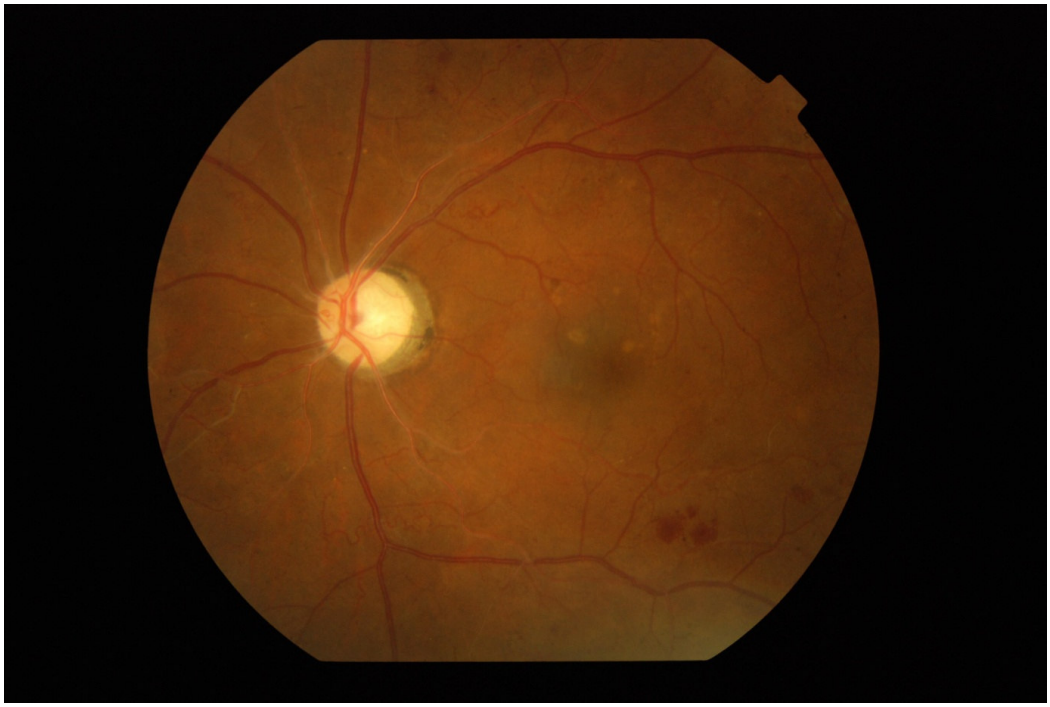

RESULTS

The age distribution in the study group was more or less even, with the patients in the age group 30 – 39 slightly more than the rest. Mean age of the patients with chronic kidney disease is 44.2.

Male patients formed 72% of the total patients in the study group. The average male : female ratio was 2.6:1.

Out of 100 patients 90 had CKD in various stages &10 belonged to post renal transplant group. The sample size in each grade of CKD showed more patients in stage 5 (31%), & stage 4 (21%) followed by stage 3 (19%), stage 1(10%) and stage 2 (9%).

The commonest cause of CKD was hypertension in 47% pts (52.2%) followed by both diabetes and hypertension in 30 patients (33.3%). Patients with only diabetes were 6 patients (6.7%) & with other causes (like IgA nephropathy , Analgesic nephropathy etc.,) were 7 patients (7.8%).

Blurring of vision was the most common symptom (85.4%) . Other symptoms were ocular irritation (9.8%) and 2.4% each with discharge & redness.

10% of patients were legally blind with visual acuity <6/60.

Of the 200 eyes included in the study 160 eyes had Anterior segment changes. 92 eyes (57.5%) had cataract . Of the 92 eyes 38 eyes were in Stage V group and 31 eyes belonged to Stage IV group.

In this study, 65 patients (130 eyes) belonged to less than 50 years (presenile age group). 49.3% of presenile patients had cataract.

A reduced Schirmer's value was noted in 54 eyes of the 200 eyes. The incidence of ocular surface disease in the study was 27%.

84 patients in the study had hypertension (HT alone or along with DM). 92 eyes out of 200 eyes studied showed hypertensive retinopathy. Higher grades of hypertensive retinopathy was more in advanced stages of CKD i.e 24 eyes in stage IV and 23 eyes in Stage V. Grade III hypertensive retinopathy was the most common grade of hypertensive retinopathy occurring in 43 eyes of the 92 eyes.

40 patients (80 eyes) in the study had Diabetes (DM alone or with HT).51 eyes showed Diabetic retinopathy changes of which a majority of 25 eyes(belonged to stage V disease. Moderate NPDR and Severe NPDR were the most common stages of Diabetic retinopathy with 29 eyes having either of these. Advanced diabetic eye disease was found in 4 patients all belonging to stage V CKD.

Prevalence of Diabetic retinopathy in CKD patients is significantly more when compared to diabetic patients without CKD.

31 eyes in the study showed changes in the macula. The most common sign was macular edema found in 13 eyes which had a mixed population of diabetic and hypertensive patients.

Other findings in the posterior segment included 4 eyes with raised CD ratio, 2 eyes with BRVO, 2 eyes with old CRAO and 3 eyes with disc pallor.

CONCLUSION

CKD is the end result of multiple systemic diseases or primary renal disease. During the natural course of the disease it affects multiple systems of the body including the eye. Detailed ocular examination was conducted in 100 patients in varying stages of CKD. In this study hypertension was the single main cause of CKD followed by DM and HT together.

Blurring of vision was the commonest ocular symptom. Most of the patients having complaints of blurring of vision were examined for the first time indicating the lack of knowledge about the potential ocular complications. Significant visual loss was due to cataract followed by Proliferative Diabetic retinopathy and macular edema.

Ocular findings that were present more in stage IV & stage V grades of CKD were cataract, lid edema, conjunctival pallor, hypertensive retinopathy, diabetic retinopathy, macular edema and CSME.

Most of the patients are detected in advanced stages of chronic kidney disease , when they become symptomatic . Hence it becomes imperative to institute steps to detect CKD at an early stage to institute specific therapy and retard the progression of renal disease . Eye can be used as a window to the kidney status , and detect any underlying renal compromise and institute early treatment.

Retinopathy is often asymptomatic in its early stage. Delay in diagnosis can result in significant visual loss. Optimized control of risk factors like renal disease which affect onset and progression of retinopathy should be approached through an intensive, multidisciplinary health care which can markedly reduce the incidence of visual loss.

In this study most of the patients were having their eyes examined for the first time. This shows the lack of awareness of the importance of early ocular evaluation among the patients.

This study is an attempt to assess the ocular status and complications associated with chronic kidney disease. It is intended to highlight the importance of ocular examination in all patients with chronic kidney disease irrespective of the presence of ocular symptoms so that necessary treatment or advice can be given before they cause irreversible visual impairment.

PART - III

Ocular manifestations in patients with Chronic Kidney Disease – a hospital based study

PROFORMA

Serial no. :

Name :

Age :

Sex :

Occupation :

Address :

Ocular complaints :

Renal status of patient

- Stage :
- Duration :
- Treatment details :

Diabetic status

- Type :
- Duration :
- Treatment details :

H/o Hypertension :

Ass. Systemic illness :

OCULAR EXAMINATION **RE** **LE**

Vision

Eyelids and lashes

Extraocular movements

Slit lamp examination

Tear meniscus

Conjunctiva

Cornea

Anterior chamber

Iris

Pupil

Lens

Fundus

IOP

Visual fields

Schirmer's test

INVESTIGATIONS

Bloodsugar

Fasting Post prandial

Blood urea

Serum creatinine

urine : Albumin

Sugar

Deposits

KEY TO MASTER CHART

ARMD	–	Age Related Macular Degeneration
BSK	–	Band Shaped Keratopathy
CAPD	–	Continuous Ambulatory Peritoneal Dialysis
CCC	–	Circum Corneal Congestion
Ce	–	Cells
CVA	–	Cerebrovascular Accident
DV	–	Defective vision
Ex	–	Exudate
F	–	Female
Gr	–	Grade
HD	–	Hemodialysis
HM	–	Hand Movements
Ht	–	Hypertension
IMC	–	Immature Cataract
LC	–	Lens changes
Li. Ed.	–	Lid Edema
M	–	Male
Mal	–	Malignant
MC	–	Mature Cataract
ME	–	Macular Edema

Mon	–	Months
N	–	Normal
NPDR	–	Non proliferative Diabetic Retinopathy
OHA	–	Oral Hypoglycemic Agents
P	–	Proptosis
Pal	–	Pallor
PCIOL	–	Posterior Chamber IntraOcular Lens
PCO	–	Posterior Capsule opacification
PDR	-	Proliferative Diabetic retinopathy
PED	–	Pigment Epithelial Detachment
Pin	–	Pingecula
PL	–	perception of Light
Red	–	reduced
Res	–	Restricted
Ret	–	Retinopathy
St	–	Stage
TRD	–	Tractional Retinal Detachment

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S.No.	Name	Age	Sex	Occupation	Address	Complaints	stage	Duration	treatment	Diabetes type	Duration	Treatment	hypertension	Systemic illness	BCVA	vision LE	eyelids RE	eyelids LE	EOM	conjunctiva RE	conjunctiva LE	Cornea	Cornea	Anterior chamber	Anterior chamber	Iris	Iris	pupil	pupil	Lens RE	Lens LE	Fundus RE	Fundus LE	iop	schirmerRE	schirmerLE	blood sugar	blood urea	serum creatinine	Hb	urine albumin	sugar	deposits	
1	Manonmani	55	F	coolie	Adampakkam	DV	St I	5 Mon		Type II	8	OHA	yes	Nil	6/60	6/24	N	N	N	N	Pin	N	N	N	N	N	N	N	MC	MC	view hazy	view hazy	N	N	N	156	144	5.7	8.2	1+	2+		
2	Muthu	65	M	coolie	Ennore	DV	St I	1		Type II	1	OHA	yes	Nil	4/60	3/60	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Severe NPDR + CSME	Severe NPDR + CSME	N	N	N	178	98	6.9	8.9	3+	3+		
3	M.Bharathiyar	42	M	Librarian	Perambur	DV	St V	4	HD	No	-	-	yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	LC	LC	Gr I HT ret	Gr I HT ret	N	N	N	85	40	1.3	11	1+	NIL		
4	Akbar Hussain	49	M	Clerk	Chennai	No	St IV	5	HD	No			yes	Nil	6/12	6/12	N	N	N	N	Pin	N	N	N	N	N	N	N	PCIOL	IMC	Gr I HT ret + ARMD	Gr I HT ret	N	Red	Red	134	80	3.4	8.9	NIL	NIL		
5	Shanmugam	65	M	Social worker	Thiruvallur	No	St I	5 Mon	Drugs	Type II	6	OHA	yes	Nil	6/18	6/12	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	CD RATIO 0.6	CD RATIO 0.6	N	N	N	144	88	4.6	9	2+	2+		
6	Vinoth kumar	34	M	Shop keeper	Velachery	No	St III	3	HD	No			yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	Red	Red	118	150	4.9	8	3+	NIL		
7	Sundari	35	F	House wife	Chrompet	no	St III	5	Drugs	No			yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret	Gr III HT ret	N	N	N	90	134	7.9	9	3+	NIL		
8	Venkatesan	50	M	Coolie	Teynampet	DV	St II	3	Drugs	Type II	13	OHA	yes	CVA	?PL	?PL	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret	Gr III HT ret	N	Red	Red	105	158	9.6	7.2	2+	1+		
9	C.Padmanabhan	46	M	LIC Agent	Neelangarai	No	St III	5	Drugs	No			yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr I HT ret	Gr I HT ret	N	N	N	99	69	3.3	7.2	1+	NIL		
10	Selvam	52	M	Mason	Mugappair	DV	St V	8	HD	Type II	15	OHA	yes	Nil	6/12	6/12	N	N	N	Pin		N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret + Moderate NPDR	Gr II HT ret + Moderate NPDR	N	N	N	137	108	12	7.9	1+	2+		
11	Sritharan	33	M	manager	Kodungayur	No	St I	4 Mon	Drugs	No			yes	Nil	6/9	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret	Gr II HT ret + ARMD	N	N	N	100	167	2	9.3	3+	NIL		
12	Sathyamoorthy	40	M	coolie	Mylapore	No	St I	7 Mon	Drugs	No			yes	Nil	6/6	6/6	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret	Gr II HT ret	N	N	N	115	158	8.1	7	3+	NIL		
13	Veeramuthu	51	M	coolie	Cuddalore	DV	St II	5 Mon	Drugs	No			Mal. HT	Nil	6/60	6/36	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr IV HT ret	Gr IV HT ret	N	N	N	87	73	3	9	1+	NIL		
14	Shanmuga sundaram	67	M	coolie	Velachery	DV	St IV	4	HD	No			yes	Nil	6/18	6/18	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	IMC	MC	Gr I HT ret + ARMD	media hazy	N	N	N	104	148	8.9	7.5	3+	TRACE		
15	Karl marx	30	M	teacher	Dharmapuri	DV	St II	2	Drugs	No			yes	Nil	6/6	2/60	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret	Gr III HT ret + macular hole	N	N	N	78	167	17	10	2+	NIL		
16	seetha lakshmi	37	F	flower vendor	Chennai	No	St IV	7	HD	no			yes	Nil	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret	Gr III HT ret + BRVO	N	N	N	111	182	16	9.7	2+	NIL		
17	Prakash	20	M	coolie	Vedaranyam	DV	St V	5	HD	no			no	? Alports	6/12	6/12	N	N	N	N	Pin	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	90	199	12	8.5	3+	NIL	1-3pus cells	
18	Lakshmi narayanan	45	M	coolie	Chennai	no	St III	4	HD	no			yes	Nil	6/18	6/6	N	N	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	Gr I HT ret	Gr I HT ret	N	N	N	94	140	6.7	10	2+	NIL		
19	Lakshmi	48	F	House wife	Washermanpet	No	St I	1	Drugs	no			yes	Nil	6/6	6/9	N	N	N	Pal	Pal	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret	Gr III HT ret	N	N	N	105	189	6.3	6.1	2+	NIL		
20	Abdul rahim	31	M	coolie	Chennai	No	St II	7	Drugs	type I	8	insulin	yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret	Gr II HT ret	N	N	N	203	40	1.5	10	1+	2+		
21	Kalpana	23	F	un employed	Nellai	No	St V	5	HD	No			yes	Nil	6/9	6/6	N	N	N	N	Pin	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret + ME	Gr III HT ret	N	N	N	92	308	19	7.1	3+	NIL		
22	Puviarasi	25	F	un employed	Coimbatore	DV	St IV	3	HD	No			yes	Nil	6/18	6/18	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret + ME	Gr III HT ret + ME	N	N	N	127	160	8.4	8	nil	NIL		
23	Neethirajan	50	M	coolie	Chennai	DV	St I	4	Drugs	Type II	10	OHA	yes	Nil	2/60	1/60	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	PDR with TRD	PDR with TRD	N	Red	Red	225	67	8.9	7	2+	3+		
24	Prabhu	26	M	coolie	nagarkoil	No	St V	5	HD	no			yes		6/6	6/6	N	N	N	Pal	Pal	N	N	N	N	N	N	N	PCIOL	PCIOL	PDR with TRD	PDR	N	N	N	165	113	7.9	6.3	3+	NIL		
25	Neela	52	F	House wife	Thiruvallur	No	St III	3	Drugs	Type II	2	OHA	yes	Nil	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Macular PED	Macular PED	N	N	N	167	145	4.7	8.9	NIL	NIL		
26	Subramani	34	M	coolie	Krishnagiri	No	St III	3	HD	no			yes		6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret	Gr II HT ret	N	Red	Red	143	56	6	9	1+	NIL		
27	Metilda	54	F	House wife	Chennai	DV	St II	1	HD	Type II	25	insulin	yes		PL	6/24	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	MC	PCIOL	No view	Vitreous hemorrhage	N	Red	Red	166	171	7.1	9.6	2+	2+		
28	Kasthuri	55	F	House wife	Adyar	Irr	St III	3	HD	Type II	8	OHA	yes	Nil	6/9	6/12	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	NAD	NAD	N	N	N	134	113	4.1	8.9	1+	1+		
29	Thomas varghese	31	M	un employed	Ambattur	No	St II	3	Drugs	No			yes		6/6	6/6	N	N	N	Pal	Pal	N	N	N	N	N	N	N	CLEAR	CLEAR	ARMD	ARMD	N	N	N	78	24	0.7	7.4	1+	NIL		
30	Josephine	38	F	House wife	Meenjur	No	St IV	4 Mon	HD	No			yes		6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret	Gr III HT ret	N	Red	Red	89	98	3.2	7.5	1+	NIL		
31	Vishwanathan	36	M	Clerk	Kanchipuram	No	St IV	6	HD	No			yes		6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret	Gr III HT ret	N	Red	Red	101	136	6	6.9	1+	NIL		
32	Venkatesh	50	M	labourer	Chennai	DV	St I	6 Mon	Drugs	Type II	3	OHA	no	Nil	6/18	6/18	N	N	N	Pal	Pal	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret	Gr III HT ret	N	Red	Red	140	81	3.2	6.2	1+	2+		
33	Thirunavukarasu	33	M	Clerk	Vellore	No	St II	1	Drugs	No			Mal. HT	Nil	6/12	6/12	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret	Gr III HT ret	N	N	N	123	66	4	8.2	1+	NIL		
34	Karthika	26	F	House wife	Tanjore	Red	St III	3	Drugs	No			yes	Nil	6/36	6/12	N	N	N	N	N	BSK	N	N	N	N	N	N	MC	IMC	No view	Gr III HT ret	N	N	N	90	177	4.1	8.7	2+	NIL		
35	Parvathy	55	F	House wife	Kanchipuram	No	St III	1	HD	No			yes		6/24	6/24	N	N	N	N	N	N	N	N	N	N	N	N	IMC	MC	Gr I HT ret + ARMD	media hazy	N	Red	Red	111	93	3.6	9.8	2+	NIL		
36	Lakshmi	38	F	Tailor	Thiruvotiyur	No	St I	3	Drugs	No			yes	Nil	6/6	6/6	N	N	N	Pal	Pal	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr I HT ret	Gr I HT ret	N	Red	Red	116	67	2	7	2+	NIL		
37	Patchai perumal	45	M	Salesman	Thoothukudi	Irr	St III	3	HD	Type II	15	OHA	yes		PL	6/18	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Vitreous hemorrhage	Gr III HT ret + PDR	N	Red	Red	155	49	3	9.1	2+	NIL		
38	Mahendran	49	M	labourer	Cholavaram	No	St I	2	HD	No			yes	Nil	6/6	6/6	N	N	N	Pin		N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret	Gr II HT ret	N	N	N	122	23	2	7	1+	NIL		
39	Visalatchi	52	F	House wife	Manali	No	St III	3	HD	No			yes		6/12	6/12	N	N	N	Pal	Pal	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret	Gr III HT ret	N	N	N	133	295	5.9	7	3+	NIL		
40	Jawahar	40	M	pvt. Company	Mangadu	DV	St V	2	HD	Type II	15	insulin	yes	Nil	6/9	6/18	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr II HT ret + Severe NPDR	Gr II HT ret + Severe NPDR + CSME	N	N	N	198	90	3.8	11	1=	2+	1-3 PUS CELLS	
41	Sivaraman	49	M	pvt. Company	Kumbakonam	No	St III	2	HD	No			yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	LC	LC	Gr II HT ret	Gr II HT ret	N	N	N	133	58	4.6	8.2	1+	NIL		
42	Suresh	23	M	un employed	Teynampet	No	St V	6 Mon	HD	No			No	IGA nephropa	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	NAD	NAD	N	N	N	62	169	8.7	8	2+	NIL		
43	Murugan	45	M	pvt. Company	Kanchipuram	No	St II	2	Drugs	No			yes		6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	Red	Red	116	52	6	8.2	1+	NIL		
44	Gokulavaradha	25	M	pvt. Company	Salem	No	PT	4	Drugs	No			No	glomeruloneg	6/12	6/9	N	N	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	ME	ME	N	N	N	120	28	1.2	7.8	NIL	NIL		
45	Mukthyar	39	M	CLERK	mannadi	DV	St IV	3	HD	type I	25	insulin	yes	Nil	6/18	6/12	Li. Ed	Li. Ed	N	N	N	BSK	N	N	N	N	N	N	IMC	IMC	PDR	PDR	N	Red	Red	120	312	17	8	4+	NIL	rbc 2+	
46	syed Moosa	30	M	pvt. Company	vyasarpadi	No	St III	3	Drugs	No			yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	ME	ME	N	N	N	89	33	2	7.9	1+	NIL		
47	Ramesh	36	M	pvt. Company	Periyapalayam	No	St IV	4	HD	No			yes		6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret + BRVO	Gr III HT ret	N	Red	Red	68	181	10	10	3+	NIL		
48	Arumugam	39	M	pvt. Company	Thiruvallur	No	PT	3	Drugs	Type II	11	insulin	yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret+ Moderate NPDR	Gr III HT ret+ Moderate NPDR	N	N	N	208	138	14	9	1+	2+		
49	Asai thambi	45	M	labourer	Thiruvannamalai	No	St V	4 Mon	HD	No			yes	Nil	6/12	6/12	N	N	N	Pal	Pal	N	N	N	N	N	N	N	PCIOL	PCIOL	Severe NPDR + CSME	Severe NPDR + CSME	N	N	N	201	78	6.2	9.1	2+	TRACE		
50	theivasigamani	64	M	labourer	Chennai	DV	St V	4	CAPD	Type II	20	insulin	yes		6/60	6/60	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret + ME	Gr III HT ret + ME	N	N	N	247	132	4.1	9.8	3+	2+		
51	Antony	51	M	Clerk	Theni	DV	St V	1	HD	No			yes		HM	6/36	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	? OLD CRAO	? OLD CRAO	N	Red	Red	156	129	4.3	9.3	2+	NIL		
52	Jayakanthan	67	M	retd teacher	Red hills	DV	St V	6 Mon	HD	Type II	15	OHA	yes	Nil	6/24	6/24	Li. Ed	Li. Ed	N	Pal	Pal	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr I HT ret	Gr I HT ret	N	Red	Red	123	102	4.9	6.8	3+	NIL	NIL	
53	Gangammal	60	F	House wife	Chennai	DV	St IV	4	HD	No			yes	Nil	6/36	6/60	N	N	N	Pal	Pal	N	N	N	N	N	N	N	MC	MC	media hazy	media hazy	N	Red	Red	252	148	7.1	6.3	3+	NIL		
54	Kuppusamy	62	M	labourer	Thiruvallur	DV	St IV	5 Mon	HD	Type II	12	insulin	yes	Nil	6/24	6/24	N	N	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	Gr III HT ret + Disc Pal + PCO	Gr III HT ret + Disc Pal + PCO+mod NP	N	N	N	61	128	5.3	10	NIL	1+		
55	John	20	M	student	Chennai	No	St IV	5 Mon	HD	No			yes	Nil	6/6	6/6	N	N	N	Pal	Pal	N	N	N	N	N	N	N	IMC	IMC	NAD	NAD	N	N	N	87	189	20	6.8	3+	NIL		
56	Muthukumar	24	M	un employed	Chennai	No	St V	5 Mon	HD	No			yes	Nil	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	old Choroiditis patch	NAD	N	N	N	100	138	9.5	6.5	3+	TRACE		
57	Kanaga	62	F	House wife	Thirunelvelli	No	St IV	4 Mon	HD	no			No	Analgesic nep	6/12	6/12	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	124	140	5.9	9.5	3+	NIL		
58	Abalagan	45	M	Clerk	Chennai	No	St III	5 Mon	Drugs	No			yes	Nil	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret	Gr III HT ret	N	N	N	59	98	3.8	9	2+	NIL		
59	Venkatesan	31	M	peon	Chennai	No	St IV	1	HD	No			no	nephrotic syn	6/9	6/6	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	IMC	IMC	NAD	NAD	N	N	N	257	197	15	8.9	4+	NIL		
60	Dalton	48	M	labourer	Kanchipuram	No	PT	1yr 6m	Drugs	No			yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	LC	LC	Gr II HT ret	Gr II HT ret	N	N	N	134	142	3.5	7.2	2+	NIL	rbc 2+	
61	Thangamani	46	F	House wife	Chennai	DV	St V	3	HD	Type II	3	OHA	No	Nil	6/18	6/18	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Severe NPDR	Severe NPDR	N	N	N	76	108	4.3	9.2	2+	3+		
62	Aruna kumari	39	F	House wife	Chennai	No	St II	6 Mon	Drugs	No			yes	Nil	6/9	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	83	180	9.3	10	2+	NIL		
63	gomathy	65	F	House wife	Cheyyar	DV	St V	2	HD	Type II	18	insulin	yes	Nil	HM	6/60	Li. Ed	Li. Ed	N	Pal	Pal	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	78	201	6.2	7.2	2+	3+		
64	thenmozhi	56	F	House wife	perambalur	DV	St III	1	CAPD	Type II	20	insulin	No	Nil	PL	6/60	N	N	N	Pal	Pal	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret + Severe NPDR	Gr III HT ret + Severe NPDR	N	N	N	178	198	5.7	8.9	1+	2+		
65	manivannan	69	M	retd clerk	chengalpattu	DV	St IV	1	HD	Type II	11	OHA	yes		6/36	6/24	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	view hazy	Optic atrophy	N	N	N	123	132	6.1	8.4	2+	NIL		
66	kalivanan	57	M	farmer	Meenjur	No	St V	1	HD	Type II	14	OHA	No	Nil	6/18	6/18	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	moderate NPDR	Moderate NPDR	N	N	N	205	169	6.7	5	2+	3+		
67	arul doss	32	M	un employed	chennai	No	St IV	1	HD	type I	24	insulin	No	Nil	6/6	6/6	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	IMC	IMC	moderate NPDR	Moderate NPDR	N	N	N	154	117	5.3	9.4	2+	2+		
68	Arokiyasamy	61	M	retd govt. servant	Chennai	DV	St V	3	HD	Type II	20	insulin	yes	Nil	6/24	6/60	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Severe NPDR	Severe NPDR + ME	N	N	N	166	160	27		2+	NIL		

S.No.	Name	Age	Sex	Occupation	Address	Complaints	stage	Duration	treatment	Diabetes type	Duration	Treatment	hypertension	Systemic illness	BCVA	vision LE	eyelids RE	eyelids LE	EOM	conjunctiva RE	conjunctiva LE	Cornea	Cornea	Anterior chamber	Anterior chamber	Iris	Iris	pupil	pupil	Lens RE	Lens LE	Fundus RE	Fundus RE	Fundus LE	top	schirmerRE	schirmerLE	blood sugar	blood urea	serum creatinine	Hb	urine albumin	sugar	deposits	
69	Jayaraman	59	M	Govt servant	Chennai	No	St IV	1	HD	Type II	14	insulin	yes	Nil	6/12	6/12	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	moderate NPDR	Moderate NPDR	N	N	N	243	99	5.2	9	NIL	NIL	PUS 2+	
70	palaninathan	65	M	business	kovilpatti	DV	St III	2	CAPD	Type II	13	insulin	yes	Nil	6/60	6/60	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	IMC	IMC	PDR with TRD	PDR	N	Red	Red	167	125	5.6	9.4	2+	2+		
71	Balakrishnan	65	M	retd govt. servant	Porur	DV	St V	2	HD	Type II	22	insulin	yes		HM	PL	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	PDR	PDR	N	Red	Red	233	115	70		3+	2+		
72	afzal	65	M	retd.	thirunindravur	DV	St V	3	HD	Type II	32	insulin	yes		PL	PL	N	N	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	Vitreous hemorrhage	Vitreous hemorrhage	N	Red	Red	304	139	39		3+	4=		
73	Komala	40	F	House wife	Chennai	No	St IV	2	HD	No			yes	Nil	6/24	6/24	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret+ Moderate NPDR	GrII HT ret + Severe NPDR + ARMD	N	Red	Red	272	191	5.7	8.9	3+	NIL		
74	Devashayam	56	M	labourer	Chennai	DV	St IV	2	HD	Type II	12	insulin	no	Nil	6/24	6/24	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	PDR	Vitreous hemorrhage	N	Red	Red	219	118	70	9	2+	NIL		
75	Chinnaraj	62	M	labourer	Chennai	DV	St V	2	HD	No			yes	Nil	6/18	6/18	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret	Gr III HT ret	N	N	N	92	227	9.3	9	4+	NIL		
76	Murugesan	46	M	labourer	Red hills	No	St IV	1	HD	Type II	3	OHA	yes	Nil	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	Severe NPDR	Severe NPDR	N	N	N	143	21	0.9	8.9	NIL	NIL		
77	Govardhan	26	M	un employed	Chennai	No	St V	1	HD	No			no	nephrotic syn	6/6	6/6	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	CD RATIO 0.8	CD RATIO 0.7	N	N	N	79	139	3.2	9.9	2+	NIL		
78	Srinivasan	65	M	retd	Kanchipuram	No	St IV	5	CAPD	Type II	5	insulin	yes	Nil	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	subretinal ppts in macula	subretinal ppts in macula	N	N	N	139	90	3.9	7.2	2+	2+		
79	Jaya	60	F	House wife	Chennai	No	St V	5 Mon	CAPD	No			No	Analgesic nep	6/9	6/9	N	N	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	NAD	NAD	N	N	N	84	92	3.7	1.4	NIL	NIL	1-3pus cells	
80	Sumitha	20	F	student	chennai	no	PT	6 Mon	Drugs	No			No	glomerulonep	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	80	102	5	7.8	1+	NIL		
81	Syed Mohammed	30	M	labourer	Kanchipuram	No	PT	5 Mon	Drugs	no			yes	Nil	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	122	34	1.4	9.4	NIL	NIL		
82	Hari	32	M	labourer	Chennai	no	PT	4 Mon	Drugs	no			no	nephrotic syn	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	85	45	1.4	10	NIL	NIL		
83	Pushpa	60	F	labourer	Chennai	DV	St V	1	HD	Type II	8	insulin	yes	Nil	5/60	4/60	N	N	N	N	N	N	N	N	N	N	N	N	PCIOL	PCIOL	PDR	PDR	N	N	N	136	28	0.9	8.2	1+	NIL	1-2 epicells	
84	Muthaiya	39	M	labourer	Chennai	No	St V	2	HD	Type II	1	OHA	yes	Nil	6/6	6/9	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	150	99	9		3+	TRACE		
85	Egaraj	25	M	labourer	Cheyyar	No	St V	10Mon	Drugs	no			yes	IgA nephropa	6/6	6/9	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr IV HT ret	Gr IV HT ret	N	N	N	135	180	11	9.2	3+	1+		
86	Gopal	61	M	Retd	Pallavaram	No	St V	10	HD	Type II	12	insulin	yes	Nil	6/12	6/18	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	Gr III HT ret + Mild NPDR	Gr III HT ret + Mild NPDR	N	N	N	201	179	9.3	10	2+	3+		
87	Poovarasan	22	M	student	mayiladuthruai	No	St V	2	HD	Type II	15	insulin	yes		6/24	6/36	N	N	N	N	N	N	N	N	N	N	N	N	IMC	IMC	PDR	PDR	N	Red	Red	189	156	7.8		3+	3+		
88	Krishnaraj	49	M	Clerk	Mylapore	No	PT	1	Drugs	No			yes	Nil	6/12	6/36	N	N	N	N	N	N	N	N	N	N	N	N	IMC	MC	tesselated fundus	media hazy	N	N	N	140	38	1.9	9	NIL	NIL		
89	Julian	41	M	worker	koratur	No	PT	2	Drugs	Type II	2	OHA	yes	Nil	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr I HT ret	Gr I HT ret	N	N	N	167	56	19	8	1+	2+		
90	Bhaskar	39	M	Clerk	Ashok nagar	No	St III	6 Mon	HD	No			yes	Nil	6/9	6/6	Li. Ed	Li. Ed	N	Pal	Pal	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret	Gr II HT ret	N	Red	Red	112	178	5.9	7.8	3+	NIL		
91	Viswanathan	60	M	labourer	Chennai	DV	St V	5 Mon	HD	Type II	12	OHA	yes	CAD, mucorm	2/60	PL	N	Li. Ed,	Res	N	N	N	N	N	N	N	N	N	IMC	IMC	media hazy	media hazy	N	N	N	98	167	6.8	7.3	2+	3+		
92	Mustafa	36	M	labourer	Chennai	No	St V	6 Mon	HD	No			yes	Nil	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	129	161	11	9.3	1+	1+		
93	Nithya	22	F	student	chennai	No	St III	2	HD	No			yes	Nil	6/6	6/6	N	N	N	Pal	Pal	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr III HT ret	Gr III HT ret	N	N	N	50	67	3.3	6.2	1+	NIL		
94	vasanthi	45	F	House wife	chennai	No	St V	2	HD	No			yes	CAHD	6/12	6/12	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret	Gr II HT ret	N	Red	Red	134	67	6.7	7.1	2+	NIL		
95	kalyani	21	F	student	Chennai	DV	St V	1	HD	no			no	glomerulonep	6/24	6/24	Li. Ed	Li. Ed	N	N	N	N	N	N	N	N	N	N	IMC	IMC	ME	ME	N	Red	Red	132	109	8.8	5.5	2+	TRACE		
96	Ezhilarasi	52	F	House wife	Kanchipuram	No	St III	1	Drugs	No			yes	Nil	6/18	6/18	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	Gr II HT ret	Gr II HT ret	N	N	N	78	76	5.4	7.2	2+	NIL		
97	Paneerselvam	45	M	labourer	chennai	DV	St IV	2	HD	no			yes		6/36	6/24	N	N	N	Pin		N	N	N	N	N	N	N	IMC	IMC	severe NPDR	Severe NPDR + ME	N	N	N	142	170	14	7.1	3+	NIL		
98	Sudhakar	51	M	labourer	Pallakad	Irr	St V	4 Mon	HD	No			yes	Nil	6/18	6/12	N	N	N	Pin		N	N	N	N	N	N	N	IMC	IMC	Gr II HT ret	Gr II HT ret	N	N	N	62	180	9.2	7.6	1+	NIL		
99	Arasu	26	M	labourer	melmaruvathur	DV,pain	PT	4Mon	HD	Type II	1	OHA	yes	Nil	6/60	6/12	N	N	N	CCC	N	N	N	Ce 4	N	N	N	N	LC	LC	NAD	NAD	N	N	N	159	72	4	7	2+	3+		
100	Balakrishnan	34	M	labourer	Chennai	Irr	PT	2	Drugs	Type II	2M	OHA	yes	Nil	6/6	6/12	N	N	N	N	N	N	N	N	N	N	N	N	CLEAR	CLEAR	NAD	NAD	N	N	N	104	35	1.2	12	NIL	NIL		